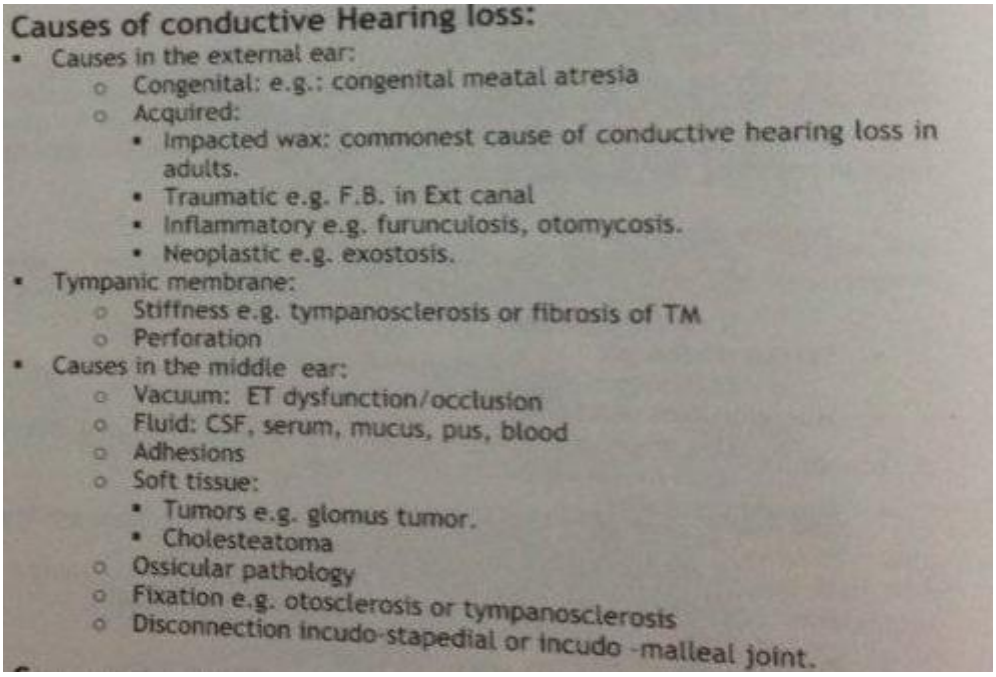
Ear

Give an account on:

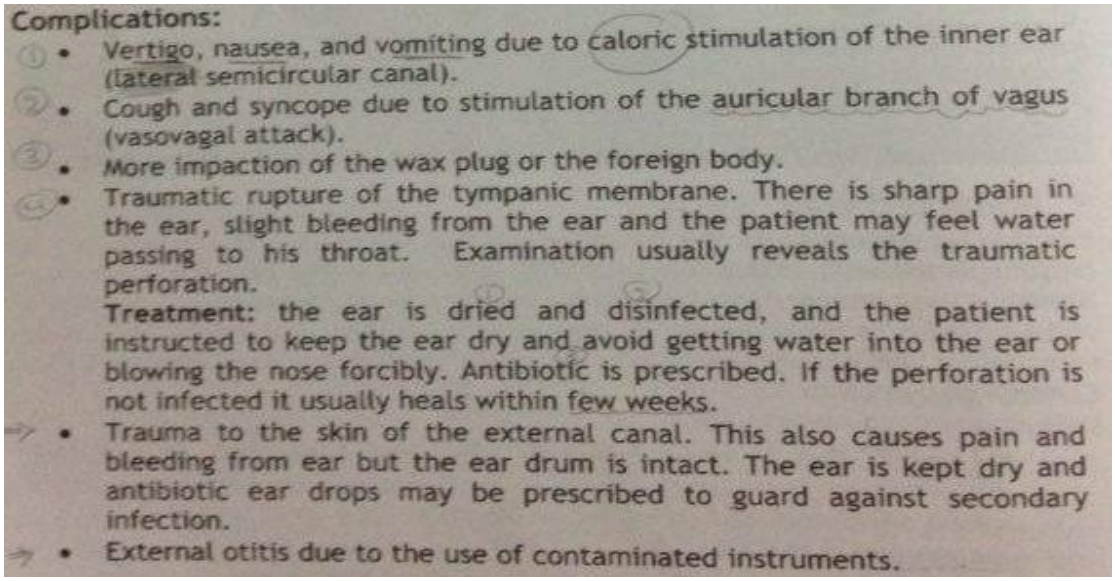
1. Etiology of conductive hearing loss (3)



Causes of conductive Hearing loss:

- Causes in the external ear:
  - Congenital: e.g.: congenital meatal atresia
  - Acquired:
    - Impacted wax: commonest cause of conductive hearing loss in adults.
    - Traumatic e.g. F.B. in Ext canal
    - Inflammatory e.g. furunculosis, otomycosis.
    - Neoplastic e.g. exostosis.
- Tympanic membrane:
  - Stiffness e.g. tympanosclerosis or fibrosis of TM
  - Perforation
- Causes in the middle ear:
  - Vacuum: ET dysfunction/occlusion
  - Fluid: CSF, serum, mucus, pus, blood
  - Adhesions
  - Soft tissue:
    - Tumors e.g. glomus tumor.
    - Cholesteatoma
  - Ossicular pathology
  - Fixation e.g. otosclerosis or tympanosclerosis
  - Disconnection incudo-stapedial or incudo -malleal joint.

2. Indications complications and contra indications of ear wash (6)



Complications:

- Vertigo, nausea, and vomiting due to caloric stimulation of the inner ear (lateral semicircular canal).
- Cough and syncope due to stimulation of the auricular branch of vagus (vasovagal attack).
- More impaction of the wax plug or the foreign body.
- Traumatic rupture of the tympanic membrane. There is sharp pain in the ear, slight bleeding from the ear and the patient may feel water passing to his throat. Examination usually reveals the traumatic perforation.
  Treatment: the ear is dried and disinfected, and the patient is instructed to keep the ear dry and avoid getting water into the ear or blowing the nose forcibly. Antibiotic is prescribed. If the perforation is not infected it usually heals within few weeks.
- Trauma to the skin of the external canal. This also causes pain and bleeding from ear but the ear drum is intact. The ear is kept dry and antibiotic ear drops may be prescribed to guard against secondary infection.
- External otitis due to the use of contaminated instruments.

Indications:

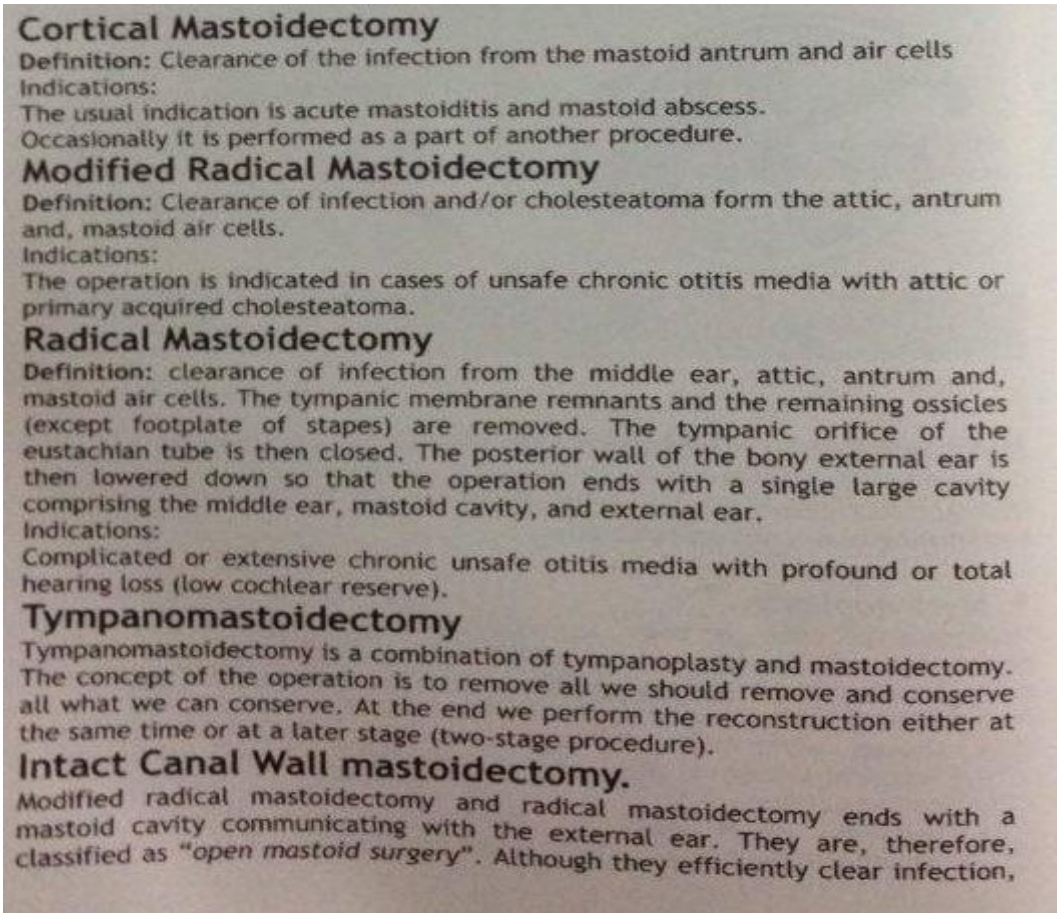
- Wax plug.
- Foreign bodies (unless it is tightly impacted in the ear canal).
- Otomycosis.

Contraindications:

- Dry perforation of the tympanic membrane to avoid infection of the middle ear.
- Recent trauma to the ear.
- Impacted foreign bodies (especially vegetables) as they may become more impacted.
- Acute external otitis as ear wash may be very painful in some cases and may even worsen the inflammation.

3. Indications of myringotomy
    - Acute suppurative otitismedia with too much bulging tympanic membrane
    - Non-resolved middle ear effusion after AOM
    - Secretory otitismedia

4. Types of mastiodectomy



Cortical Mastoidectomy

Definition: Clearance of the infection from the mastoid antrum and air cells

Indications:

The usual indication is acute mastoiditis and mastoid abscess.

Occasionally it is performed as a part of another procedure.

Modified Radical Mastoidectomy

Definition: Clearance of infection and/or cholesteatoma form the attic, antrum and, mastoid air cells.

Indications:

The operation is indicated in cases of unsafe chronic otitis media with attic or primary acquired cholesteatoma.

Radical Mastoidectomy

Definition: clearance of infection from the middle ear, attic, antrum and, mastoid air cells. The tympanic membrane remnants and the remaining ossicles (except footplate of stapes) are removed. The tympanic orifice of the eustachian tube is then closed. The posterior wall of the bony external ear is then lowered down so that the operation ends with a single large cavity comprising the middle ear, mastoid cavity, and external ear.

Indications:

Complicated or extensive chronic unsafe otitis media with profound or total hearing loss (low cochlear reserve).

Tympanomastoidectomy

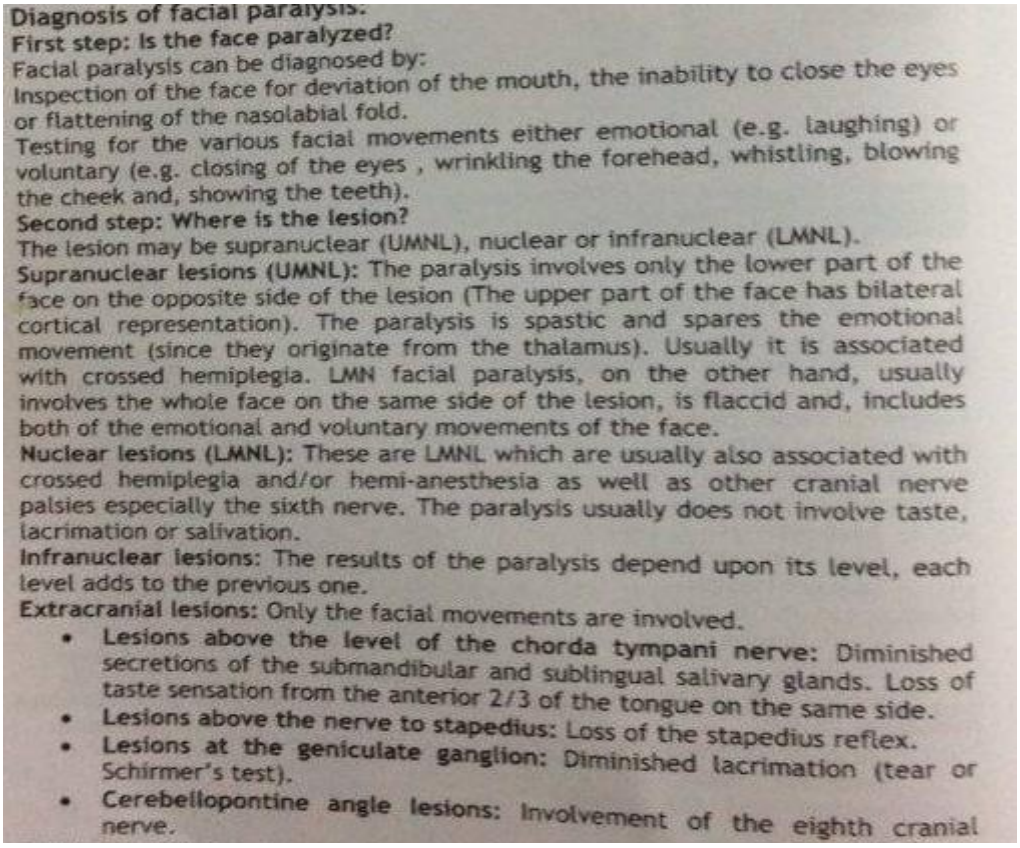
Tympanomastoidectomy is a combination of tympanoplasty and mastoidectomy. The concept of the operation is to remove all we should remove and conserve all what we can conserve. At the end we perform the reconstruction either at the same time or at a later stage (two-stage procedure).

Intact Canal Wall mastoidectomy.

Modified radical mastoidectomy and radical mastoidectomy ends with a mastoid cavity communicating with the external ear. They are, therefore, classified as "*open mastoid surgery*". Although they efficiently clear infection,

5. Treatment of menier's disease
    - Medical
        i. The acute attacks of vertigo is controlled by sedatives (diazepam) and anti-vertigo drugs (Cinnarizine and Dramamine)
        ii. In between attacks the patient is advised to use low salt diet and use diuretics (Thiazide) and antivertigo drugs
        iii. Selective ablation of the vestibular labyrinth by intra-tympanic instillation of gentamycin
    - Surgical treatment
        i. Decompression of endolymphatic sac promote absorption of endolymph
        ii. Labyrinthectomy in patients without serviceable hearing
        iii. Vestibular neurectomy in patients with serviceable hearing

6. Diagnostic features and levels of facial palsy

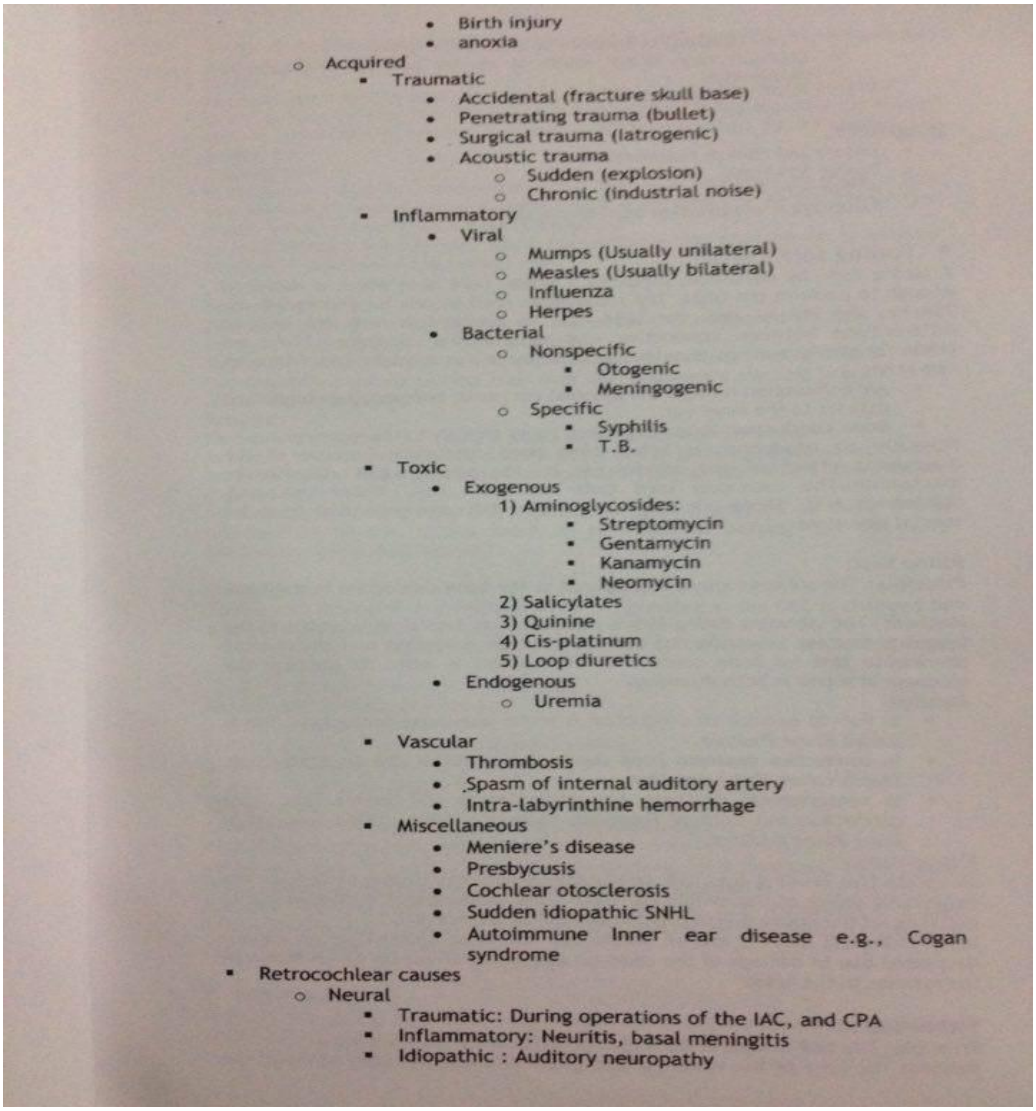


Diagnosis of facial paralysis:
First step: Is the face paralyzed?
Facial paralysis can be diagnosed by:
Inspection of the face for deviation of the mouth, the inability to close the eyes or flattening of the nasolabial fold.
Testing for the various facial movements either emotional (e.g. laughing) or voluntary (e.g. closing of the eyes , wrinkling the forehead, whistling, blowing the cheek and, showing the teeth).
Second step: Where is the lesion?
The lesion may be supranuclear (UMNL), nuclear or infranuclear (LMNL).
Supranuclear lesions (UMNL): The paralysis involves only the lower part of the face on the opposite side of the lesion (The upper part of the face has bilateral cortical representation). The paralysis is spastic and spares the emotional movement (since they originate from the thalamus). Usually it is associated with crossed hemiplegia. LMN facial paralysis, on the other hand, usually involves the whole face on the same side of the lesion, is flaccid and, includes both of the emotional and voluntary movements of the face.
Nuclear lesions (LMNL): These are LMNL which are usually also associated with crossed hemiplegia and/or hemi-anesthesia as well as other cranial nerve palsies especially the sixth nerve. The paralysis usually does not involve taste, lacrimation or salivation.
Infranuclear lesions: The results of the paralysis depend upon its level, each level adds to the previous one.
Extracranial lesions: Only the facial movements are involved.

- **Lesions above the level of the chorda tympani nerve:** Diminished secretions of the submandibular and sublingual salivary glands. Loss of taste sensation from the anterior 2/3 of the tongue on the same side.
- **Lesions above the nerve to stapedius:** Loss of the stapedius reflex.
- **Lesions at the geniculate ganglion:** Diminished lacrimation (tear or Schirmer's test).
- **Cerebellopontine angle lesions:** Involvement of the eighth cranial nerve.

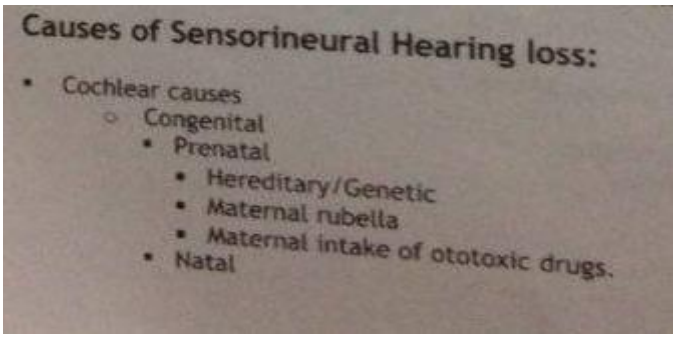
7. Types of mastoid abscess
8. Clinical picture of secretory otitis media
    - Symptoms
        i. Hearing loss
        ii. Feeling of blockage
        iii. Tinnitus
        iv. Pain (in acute cases)
    - Examination
        i. With otoscopy (Air bubbles – Hair line – Opaque TM – Retracted tympanic membrane)
        ii. Seiglization : restricted mobility of TM
        iii. TFT:Conductive hearing loss
        iv. Audiometry: Conductive HL
        v. Tympanometry : Type B
        vi. Absent reflexes

9. Causes of perceptive deafness



- Birth injury
- anoxia

- Acquired
  - Traumatic
    - Accidental (fracture skull base)
    - Penetrating trauma (bullet)
    - Surgical trauma (iatrogenic)
    - Acoustic trauma
      - Sudden (explosion)
      - Chronic (industrial noise)
  - Inflammatory
    - Viral
      - Mumps (Usually unilateral)
      - Measles (Usually bilateral)
      - Influenza
      - Herpes
    - Bacterial
      - Nonspecific
        - Otogenic
        - Meningogenic
      - Specific
        - Syphilis
        - T.B.
  - Toxic
    - Exogenous
      1) Aminoglycosides:
         - Streptomycin
         - Gentamycin
         - Kanamycin
         - Neomycin
      2) Salicylates
      3) Quinine
      4) Cis-platinum
      5) Loop diuretics
    - Endogenous
      - Uremia
  - Vascular
    - Thrombosis
    - Spasm of internal auditory artery
    - Intra-labyrinthine hemorrhage
  - Miscellaneous
    - Meniere's disease
    - Presbycusis
    - Cochlear otosclerosis
    - Sudden idiopathic SNHL
    - Autoimmune Inner ear disease e.g., Cogan syndrome
- Retrocochlear causes
  - Neural
    - Traumatic: During operations of the IAC, and CPA
    - Inflammatory: Neuritis, basal meningitis
    - Idiopathic : Auditory neuropathy



Causes of Sensorineural Hearing loss:

- Cochlear causes
  - Congenital
    - Prenatal
      - Hereditary/Genetic
      - Maternal rubella
      - Maternal intake of ototoxic drugs.
    - Natal

10. Clinical picture of unsafe CSOM
    - Symptoms
      i. Otorrhea (scanty – purulent – offensive – continuous)
      ii. Tinnitus
      iii. Hearing loss
        - Minimal early in disease

- Severe when there is ossicular pathology or cochlear involvement

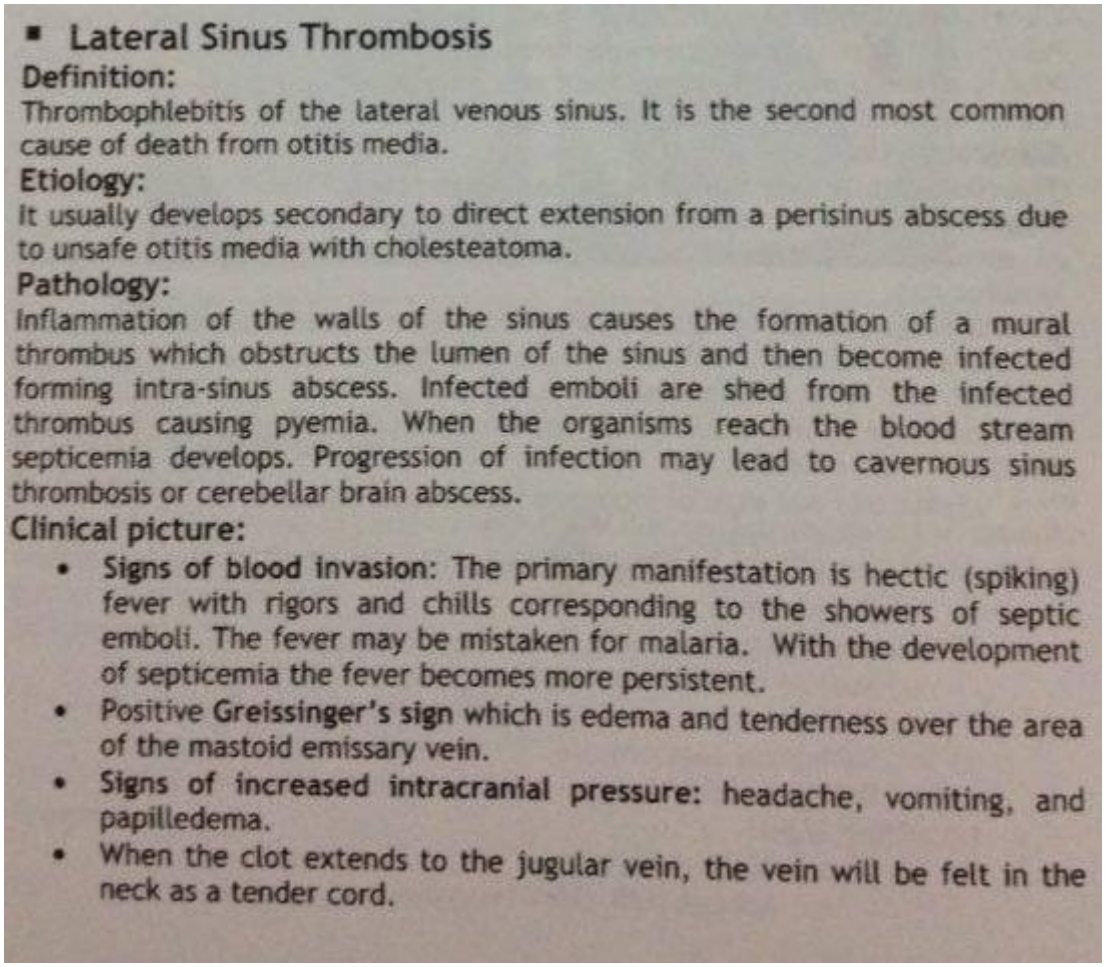
+/- symptoms of complications

- Signs
  i. Discharge
  ii. Perforation (in pars flacida in 1ry and marginal in 2ndry
  iii. Hyperemic polypi
  iv. Granulation tissue and cholesteatoma
- TFT
  i. Conductive hearing loss
  ii. Rinne's negative in diseased ear
  iii. Weber lateralizes to diseased ear
  iv. Can show mixed hearing loss pattern

11. Enumerate complications of otitis media and clinical picture of lateral sinus thrombo phlebitis.(4)

- Cranial
  i. Acute mastoiditis and mastoid abscess
  ii. Petrositis
  iii. Labyrinthitis
  iv. Facial paralysis
  v. Osteomyelitis of the temporal bone
- Intracranial complications
  i. Extradural abscess
  ii. Meningitis
  iii. Subdural abscess
  iv. Brain abscess
  v. Temporal lobe abcess
  vi. Cerebellar abscess
  vii. Lateral sinus thrombophlebitis
  viii. Otitis hydro cephalus
- Extraceanial
  i. External otitis

ii. Cervical lymphadenitis



- **Lateral Sinus Thrombosis**

Definition:
Thrombophlebitis of the lateral venous sinus. It is the second most common cause of death from otitis media.

Etiology:
It usually develops secondary to direct extension from a perisinus abscess due to unsafe otitis media with cholesteatoma.

Pathology:
Inflammation of the walls of the sinus causes the formation of a mural thrombus which obstructs the lumen of the sinus and then become infected forming intra-sinus abscess. Infected emboli are shed from the infected thrombus causing pyemia. When the organisms reach the blood stream septicemia develops. Progression of infection may lead to cavernous sinus thrombosis or cerebellar brain abscess.

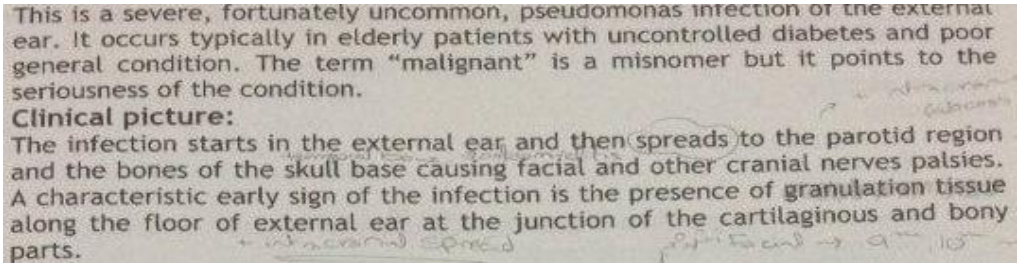
Clinical picture:

- **Signs of blood invasion**: The primary manifestation is hectic (spiking) fever with rigors and chills corresponding to the showers of septic emboli. The fever may be mistaken for malaria. With the development of septicemia the fever becomes more persistent.
- Positive **Greissinger's sign** which is edema and tenderness over the area of the mastoid emissary vein.
- **Signs of increased intracranial pressure:** headache, vomiting, and papilledema.
- When the clot extends to the jugular vein, the vein will be felt in the neck as a tender cord.

12. Investigations for dizzy patient(2)
    - Recording of nystagmus by (frenzel glass ENG oor VNG)
    - Posturography
    - Audiometric evaluation
    - Radiology: CT-MRI
    - Blood examination Immunologic testing and blood sugar
13. Types and treatment of tympanic perforation
    - Marginal – attic – central
    - Treatment
        i. Conservative if there is no evidence of ossicular disruption. The patient is asked to keep the ear absolutely dry and to avoid blowing of the nose. A short course of antibiotic may be given. Antibiotic ear drops are unnecessary

ii. Myringoplasty if perforation fails to heal within 3 months
iii. Tympanoplasty if there is ossicular disruption

14. Etiology and clinical picture of malignant otitis

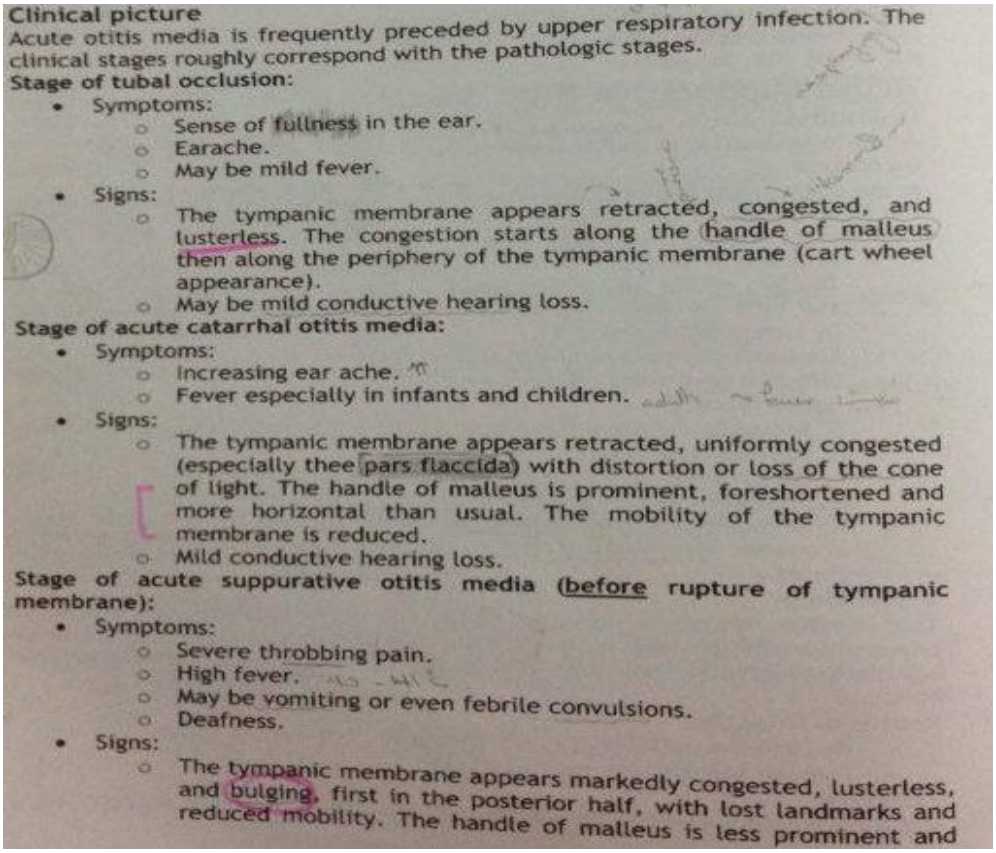

This is a severe, fortunately uncommon, pseudomonas infection of the external ear. It occurs typically in elderly patients with uncontrolled diabetes and poor general condition. The term "malignant" is a misnomer but it points to the seriousness of the condition.

Clinical picture:

The infection starts in the external ear and then spreads to the parotid region and the bones of the skull base causing facial and other cranial nerves palsies. A characteristic early sign of the infection is the presence of granulation tissue along the floor of external ear at the junction of the cartilaginous and bony parts.

15. Etiology of facial paralysis
    - Idiopathic facial nerve palsy (Bell's palsy)
    - Traumatic(birth injuries – skull base fracture – surgical trauma)
    - Inflammatory (Viral : Herpes zoster oticus – bacterial : CSOM)
    - Neoplastic(Facial nerve neuroma – malignant tumor of CPA or parotid)
    - Central : stroke
16. Clinical picture and investigations for acoustic neuroma(2)
    - PTA shows high frequency hearing loss
    - Speech audiometry shows low speech discrimination scores
    - Tympanometry shows absence or decay of the stapedius reflex
    - ABR show delayes or absent wave V
    - Radiology
        i. CT scan (erosion of IAC) and very small tumors are overlooked
        ii. MRI (best imaging tool)

17. Clinical picture of acute otitis media



Clinical picture

Acute otitis media is frequently preceded by upper respiratory infection. The clinical stages roughly correspond with the pathologic stages.

Stage of tubal occlusion:

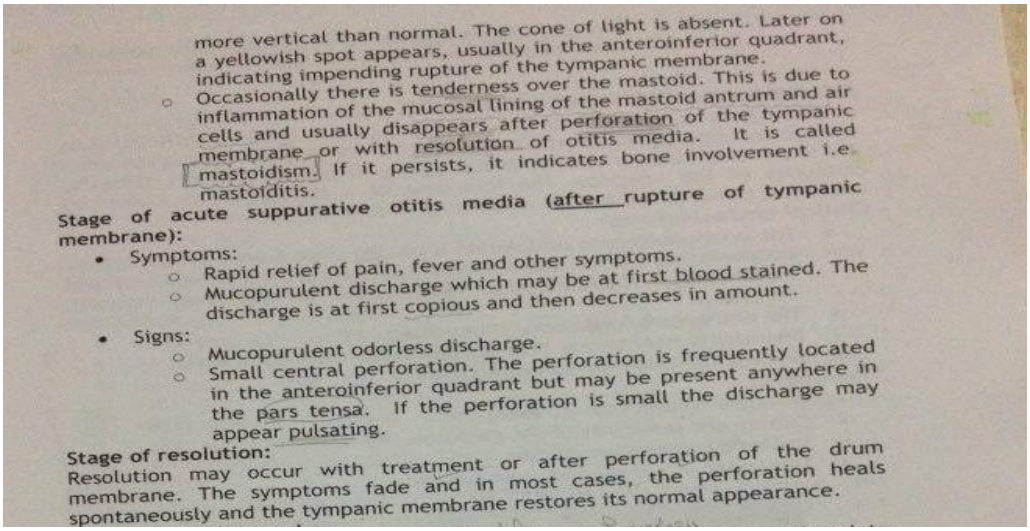
- Symptoms:
  - Sense of fullness in the ear.
  - Earache.
  - May be mild fever.
- Signs:
  - The tympanic membrane appears retracted, congested, and lusterless. The congestion starts along the handle of malleus then along the periphery of the tympanic membrane (cart wheel appearance).
  - May be mild conductive hearing loss.

Stage of acute catarrhal otitis media:

- Symptoms:
  - Increasing ear ache.
  - Fever especially in infants and children.
- Signs:
  - The tympanic membrane appears retracted, uniformly congested (especially thee pars flaccida) with distortion or loss of the cone of light. The handle of malleus is prominent, foreshortened and more horizontal than usual. The mobility of the tympanic membrane is reduced.
  - Mild conductive hearing loss.

Stage of acute suppurative otitis media (before rupture of tympanic membrane):

- Symptoms:
  - Severe throbbing pain.
  - High fever.
  - May be vomiting or even febrile convulsions.
  - Deafness.
- Signs:
  - The tympanic membrane appears markedly congested, lusterless, and bulging, first in the posterior half, with lost landmarks and reduced mobility. The handle of malleus is less prominent and



more vertical than normal. The cone of light is absent. Later on a yellowish spot appears, usually in the anteroinferior quadrant, indicating impending rupture of the tympanic membrane.

- Occasionally there is tenderness over the mastoid. This is due to inflammation of the mucosal lining of the mastoid antrum and air cells and usually disappears after perforation of the tympanic membrane or with resolution of otitis media. It is called mastoidism. If it persists, it indicates bone involvement i.e. mastoiditis.

Stage of acute suppurative otitis media (after rupture of tympanic membrane):

- Symptoms:
  - Rapid relief of pain, fever and other symptoms.
  - Mucopurulent discharge which may be at first blood stained. The discharge is at first copious and then decreases in amount.
- Signs:
  - Mucopurulent odorless discharge.
  - Small central perforation. The perforation is frequently located in the anteroinferior quadrant but may be present anywhere in the pars tensa. If the perforation is small the discharge may appear pulsating.

Stage of resolution:

Resolution may occur with treatment or after perforation of the drum membrane. The symptoms fade and in most cases, the perforation heals spontaneously and the tympanic membrane restores its normal appearance.

18. Diagnosis and treatment of secretory otitis media

- Symptoms
  - i. Hearing loss
  - ii. Feeling of blockage
  - iii. Tinnitus
  - iv. Pain (in acute cases)
- Examination

i. With otoscopy (Air bubbles – Hair line – Opaque TM – Retracted tympanic membrane)
ii. Seiglization : restricted mobility of TM
iii. TFT:Conductive hearing loss
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v. Tympanometry : Type B
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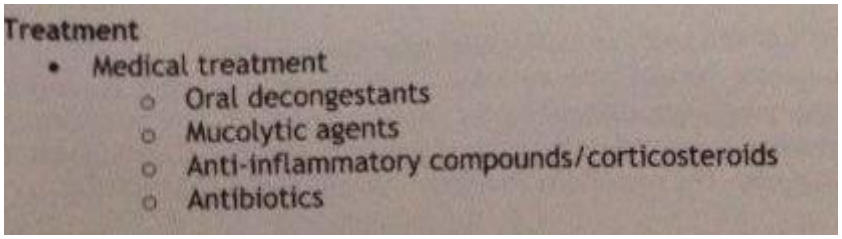
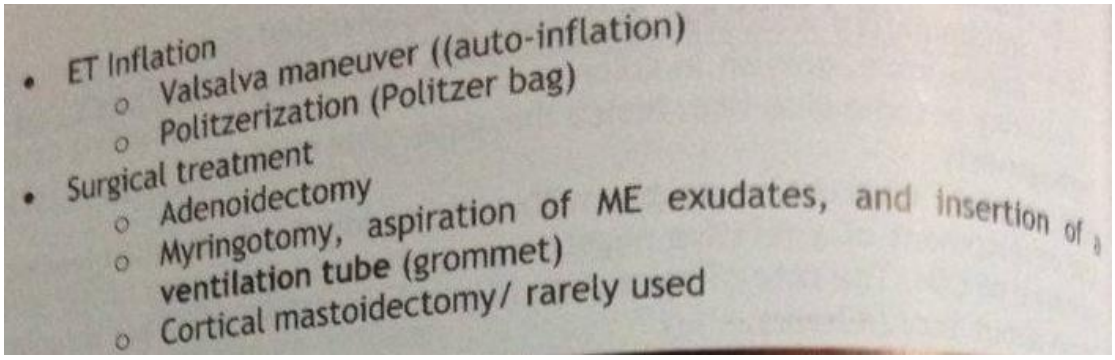




19. Etiology and diagnosis of vertigo

Etiology:

Vertigo can result from peripheral lesions involving the vestibular end organs or from central lesions involving the central vestibular pathways and centers.

- Peripheral causes:
  - Meniere's disease.
  - Motion sickness.
  - Positional vertigo.
  - Congenital: Congenital syphilis.
  - Traumatic: Skull base fractures.
  - Inflammatory: Vestibular neuritis and labyrinthitis.
  - Vestibulo toxic drugs: Gentamycin and streptomycin.
- Central causes:
  - Vascular: Vertebro-basilar insufficiency, lateral medullary syndrome.
  - Trauma: Concussion and whiplash injuries of the neck.
  - Inflammatory: Cerebellar abscess.
  - Multiple sclerosis.

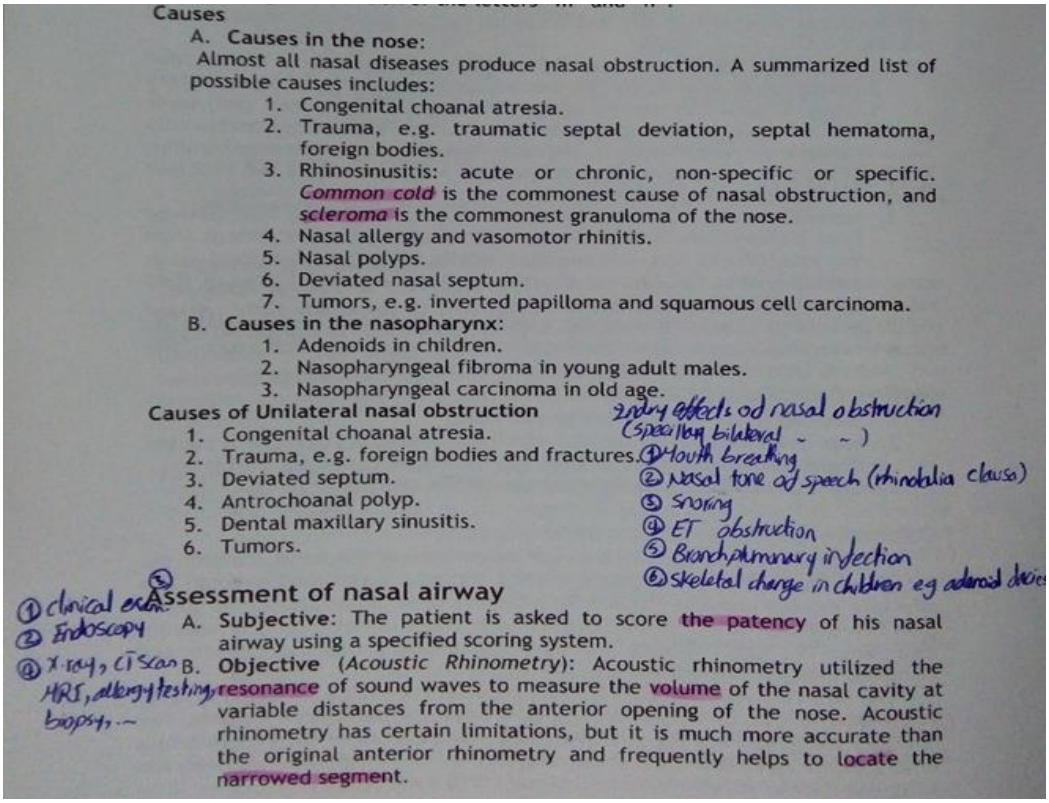
Diagnosis:

- History: Proper history taking is of prime importance and frequently provides sufficient clues for the diagnosis.
- Examination of the ear, nervous system and the eyes.
- Tests for ataxia (e.g. Romberg's test) and other cerebellar tests are done at this stage.
- Observation and recording of nystagmus:
  - Direct observation of the eyes using 15+ diopters glasses (Frenzel's glasses).
  - Electronystagmography (ENG): Electronic recording of nystagmus by measuring the corneo-retinal potentials.
  - Video-nystagmography which is a more recent version of ENG.

- Vestibular tests with recording of nystagmus:
  - Caloric test.
  - Positional and positioning tests.
  - Ocular fixation testing.
- Tests of posture:
  - Posturography.
- Audiometric evaluation.
- Radiological examination: CT and MRI.
- Other investigations e.g. blood examination and immunologic tests and blood sugar.

Nose

1. Etiology and investigation of a patient with unilateral nasal obstruction.



Causes

A. **Causes in the nose:**
Almost all nasal diseases produce nasal obstruction. A summarized list of possible causes includes:
1. Congenital choanal atresia.
2. Trauma, e.g. traumatic septal deviation, septal hematoma, foreign bodies.
3. Rhinosinusitis: acute or chronic, non-specific or specific. *Common cold* is the commonest cause of nasal obstruction, and *scleroma* is the commonest granuloma of the nose.
4. Nasal allergy and vasomotor rhinitis.
5. Nasal polyps.
6. Deviated nasal septum.
7. Tumors, e.g. inverted papilloma and squamous cell carcinoma.

B. **Causes in the nasopharynx:**
1. Adenoids in children.
2. Nasopharyngeal fibroma in young adult males.
3. Nasopharyngeal carcinoma in old age.

Causes of Unilateral nasal obstruction
1. Congenital choanal atresia.
2. Trauma, e.g. foreign bodies and fractures.
3. Deviated septum.
4. Antrochoanal polyp.
5. Dental maxillary sinusitis.
6. Tumors.

2ndry effects of nasal obstruction
(Specially bilateral ~ ~)
① Mouth breathing
② Nasal tone of speech (rhinolalia clausa)
③ Snoring
④ ET obstruction
⑤ Bronchpulmonary infection
⑥ Skeletal change in children eg adenoid face

Assessment of nasal airway

① clinical exam.
② Endoscopy
③ X-ray, CT scan, MRI, allergy testing, biopsy, ...

A. **Subjective**: The patient is asked to score the patency of his nasal airway using a specified scoring system.

B. **Objective** (*Acoustic Rhinometry*): Acoustic rhinometry utilized the resonance of sound waves to measure the volume of the nasal cavity at variable distances from the anterior opening of the nose. Acoustic rhinometry has certain limitations, but it is much more accurate than the original anterior rhinometry and frequently helps to locate the narrowed segment.

2. Enumerate complications of sinusitis.

Cranial complications

- Sinonasal
  1. Osteitis – osteomyelitis – pott's puffy tumor.
  2. Mucocele – pyocele.
  3. Periorbital and frontal fistula.
- Orbital
  1. Eyelid abcess.
  2. Orbital cellulitis.
  3. Supraorbital abcess.
  4. Orbital abcess.
  5. Optic neuritis.
- Ear
  1. Eustachian tube dysfunction.
  2. Otitis media.

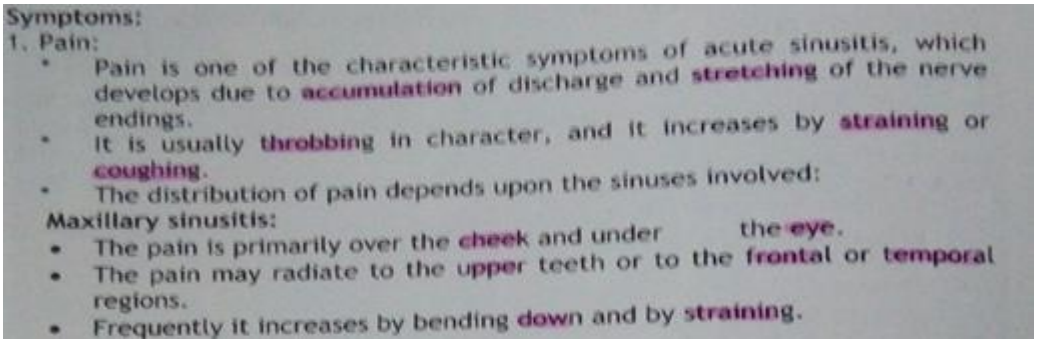
Intracranial complication

- Meningeal
  1. Meningitis
  2. Extradural abcess
  3. Subdural abcess
- Brain
  1. Encephalitis
  2. Frontal lobe abcess.
- Venous sinuses
  1. Superior saggital sinus thrombosis.
  2. Cavernous sinus thrombosis.

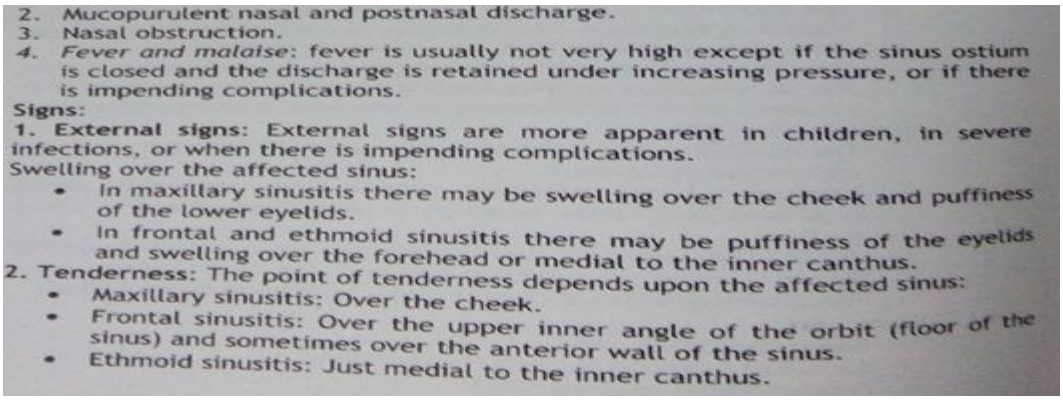
Extra-cranial

1. Ascending and descending infections.
2. Septicemia and pyemia.

3. Clinical picture of acute maxillary sinusitis.


Symptoms:
1. Pain:
- Pain is one of the characteristic symptoms of acute sinusitis, which develops due to accumulation of discharge and stretching of the nerve endings.
- It is usually throbbing in character, and it increases by straining or coughing.
- The distribution of pain depends upon the sinuses involved:

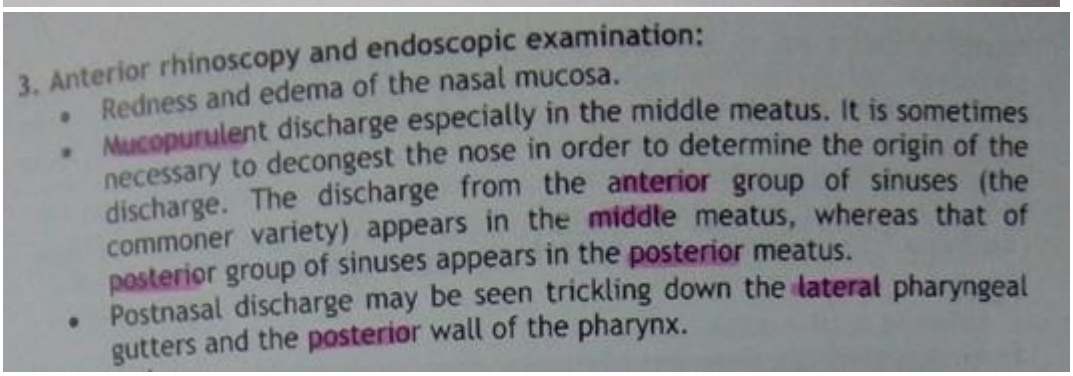
Maxillary sinusitis:
- The pain is primarily over the cheek and under the eye.
- The pain may radiate to the upper teeth or to the frontal or temporal regions.
- Frequently it increases by bending down and by straining.


2. Mucopurulent nasal and postnasal discharge.
3. Nasal obstruction.
4. *Fever and malaise*: fever is usually not very high except if the sinus ostium is closed and the discharge is retained under increasing pressure, or if there is impending complications.

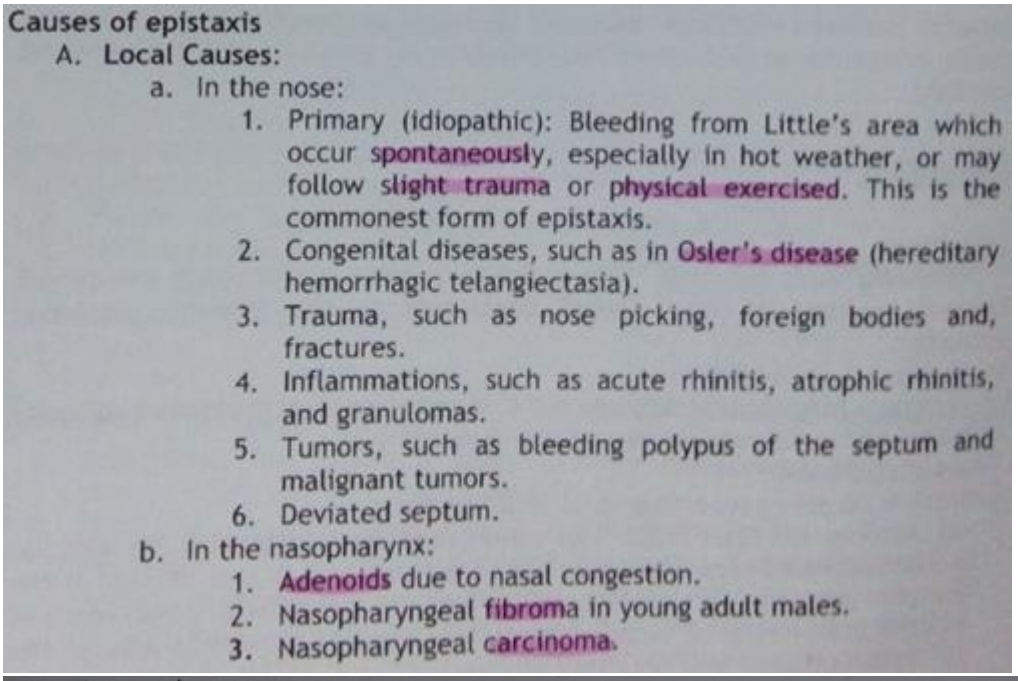
Signs:
1. **External signs**: External signs are more apparent in children, in severe infections, or when there is impending complications.

Swelling over the affected sinus:
- In maxillary sinusitis there may be swelling over the cheek and puffiness of the lower eyelids.
- In frontal and ethmoid sinusitis there may be puffiness of the eyelids and swelling over the forehead or medial to the inner canthus.

2. **Tenderness**: The point of tenderness depends upon the affected sinus:
- Maxillary sinusitis: Over the cheek.
- Frontal sinusitis: Over the upper inner angle of the orbit (floor of the sinus) and sometimes over the anterior wall of the sinus.
- Ethmoid sinusitis: Just medial to the inner canthus.


3. Anterior rhinoscopy and endoscopic examination:
- Redness and edema of the nasal mucosa.
- Mucopurulent discharge especially in the middle meatus. It is sometimes necessary to decongest the nose in order to determine the origin of the discharge. The discharge from the anterior group of sinuses (the commoner variety) appears in the middle meatus, whereas that of posterior group of sinuses appears in the posterior meatus.
- Postnasal discharge may be seen trickling down the lateral pharyngeal gutters and the posterior wall of the pharynx.

4. Enumerate causes of epistaxis – Systemic causes of epistaxis.(3)



Causes of epistaxis

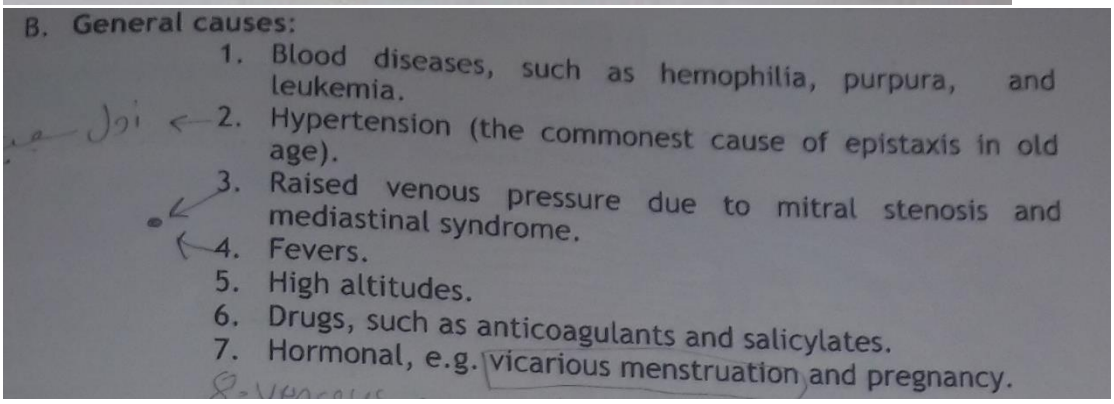
A. **Local Causes:**

a. In the nose:

1. Primary (idiopathic): Bleeding from Little's area which occur spontaneously, especially in hot weather, or may follow slight trauma or physical exercised. This is the commonest form of epistaxis.
2. Congenital diseases, such as in Osler's disease (hereditary hemorrhagic telangiectasia).
3. Trauma, such as nose picking, foreign bodies and, fractures.
4. Inflammations, such as acute rhinitis, atrophic rhinitis, and granulomas.
5. Tumors, such as bleeding polypus of the septum and malignant tumors.
6. Deviated septum.

b. In the nasopharynx:

1. Adenoids due to nasal congestion.
2. Nasopharyngeal fibroma in young adult males.
3. Nasopharyngeal carcinoma.



B. **General causes:**

1. Blood diseases, such as hemophilia, purpura, and leukemia.
2. Hypertension (the commonest cause of epistaxis in old age).
3. Raised venous pressure due to mitral stenosis and mediastinal syndrome.
4. Fevers.
5. High altitudes.
6. Drugs, such as anticoagulants and salicylates.
7. Hormonal, e.g. vicarious menstruation and pregnancy.

5. Management of epistaxis – management of severe epistaxis.

Management

A- First Aid Measures:

1. The patient is put in the sitting position, and slightly inclined forward, with his mouth open. He is instructed to breath quietly through the mouth.
2. The patient is instructed to squeeze (pinch) the ala of the nose for few minutes by his fingers. This applies direct pressure on Little's area which is the most frequent source of epistaxis.
3. Ice compresses are applied to the bridge of the nose to induce reflex vasoconstriction of the nasal mucosa.
4. A small cotton pack soaked with adrenalin or other commercial vasoconstrictor nasal drops may be placed inside the nose *unless the patient is hypertensive.*

B- Treatment in hospital:

I- General measures:

1. Measurement of blood pressure and other vital signs.
2. Anti-shock measures if required.
3. Coagulants and other medications may be given according to the diagnosed etiology.

I- Local Measures:

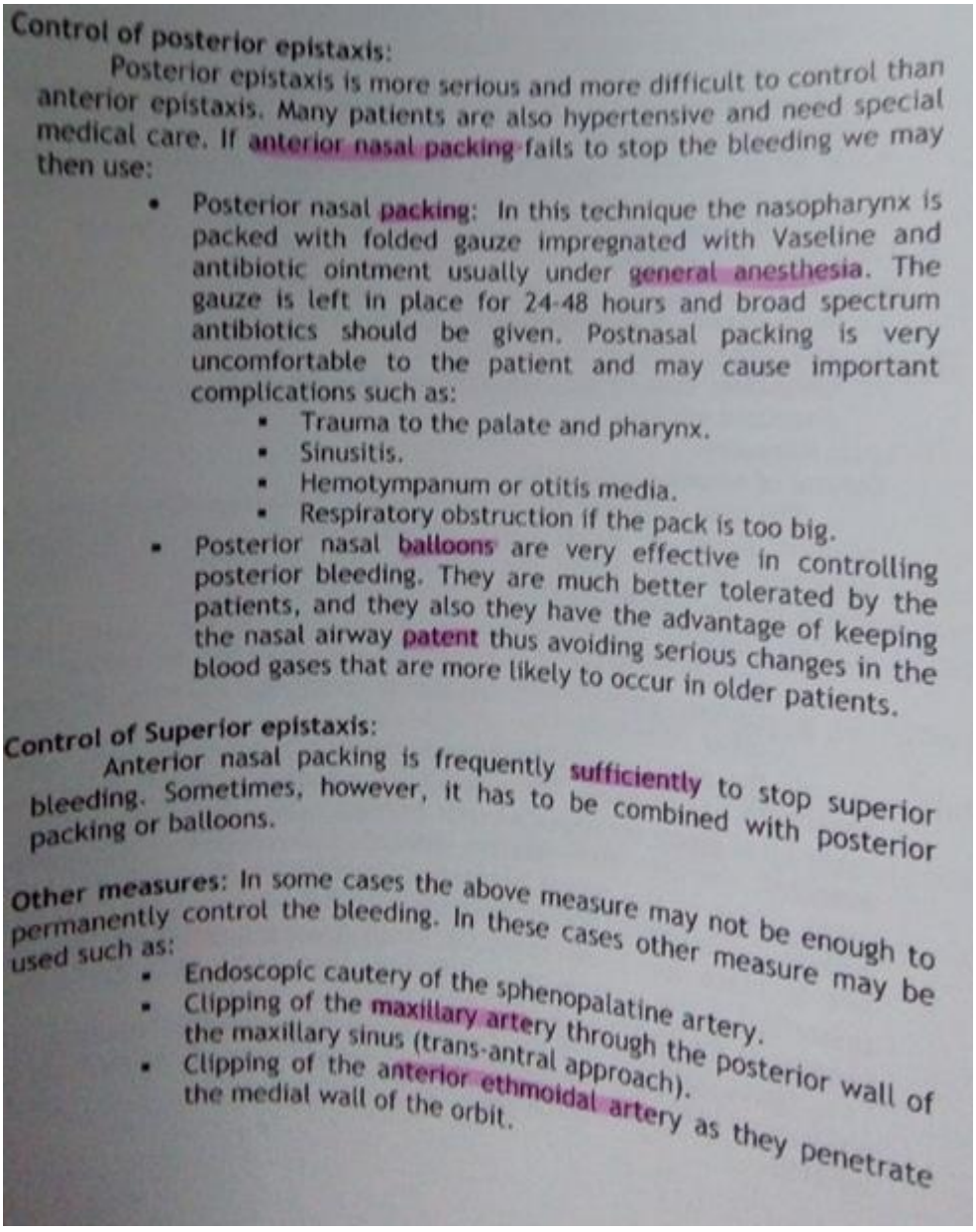
Control of anterior epistaxis:

The nose is packed for 10 minutes with pledges of cotton soaked in a solution made of equal parts of epinephrine ~~1:1000~~ and Xylocaine (lidocaine HCl) 4%. When the bleeding stops or significantly lessens we search for the bleeding point and try to seal it with cautery:

- Chemical cautery with silver nitrate sticks or 30% solution is usual enough for bleeding from Little's area especially in children.
- Galvano-cautery and coagulating diathermy are more effective in other situations.

If the above technique is unsuccessful or unavailable, an anterior nasal pack can be used. The classical time-honored anterior nasal pack is made up of gauze impregnated with Vaseline (petrolatum) and occasionally antibiotic ointment. More modern types are now available, such as Merocel packs. Some of the recent packs are also fit with an airway so that the patient can breathe through the nose with the pack in place. The pack is left in place for 24-48 hours. A broad spectrum antibiotic should be prescribed if the pack is to be left more than 24 hours to guard against sinusitis or the toxic shock syndrome.

Nasal balloons are better reserved for posterior epistaxis since anterior balloons may slough the nasal mucosa if they are erroneously over-inflated.



Control of posterior epistaxis:

Posterior epistaxis is more serious and more difficult to control than anterior epistaxis. Many patients are also hypertensive and need special medical care. If anterior nasal packing fails to stop the bleeding we may then use:

- Posterior nasal packing: In this technique the nasopharynx is packed with folded gauze impregnated with Vaseline and antibiotic ointment usually under general anesthesia. The gauze is left in place for 24-48 hours and broad spectrum antibiotics should be given. Postnasal packing is very uncomfortable to the patient and may cause important complications such as:
  - Trauma to the palate and pharynx.
  - Sinusitis.
  - Hemotympanum or otitis media.
  - Respiratory obstruction if the pack is too big.
- Posterior nasal balloons are very effective in controlling posterior bleeding. They are much better tolerated by the patients, and they also they have the advantage of keeping the nasal airway patent thus avoiding serious changes in the blood gases that are more likely to occur in older patients.

Control of Superior epistaxis:

Anterior nasal packing is frequently sufficiently to stop superior bleeding. Sometimes, however, it has to be combined with posterior packing or balloons.

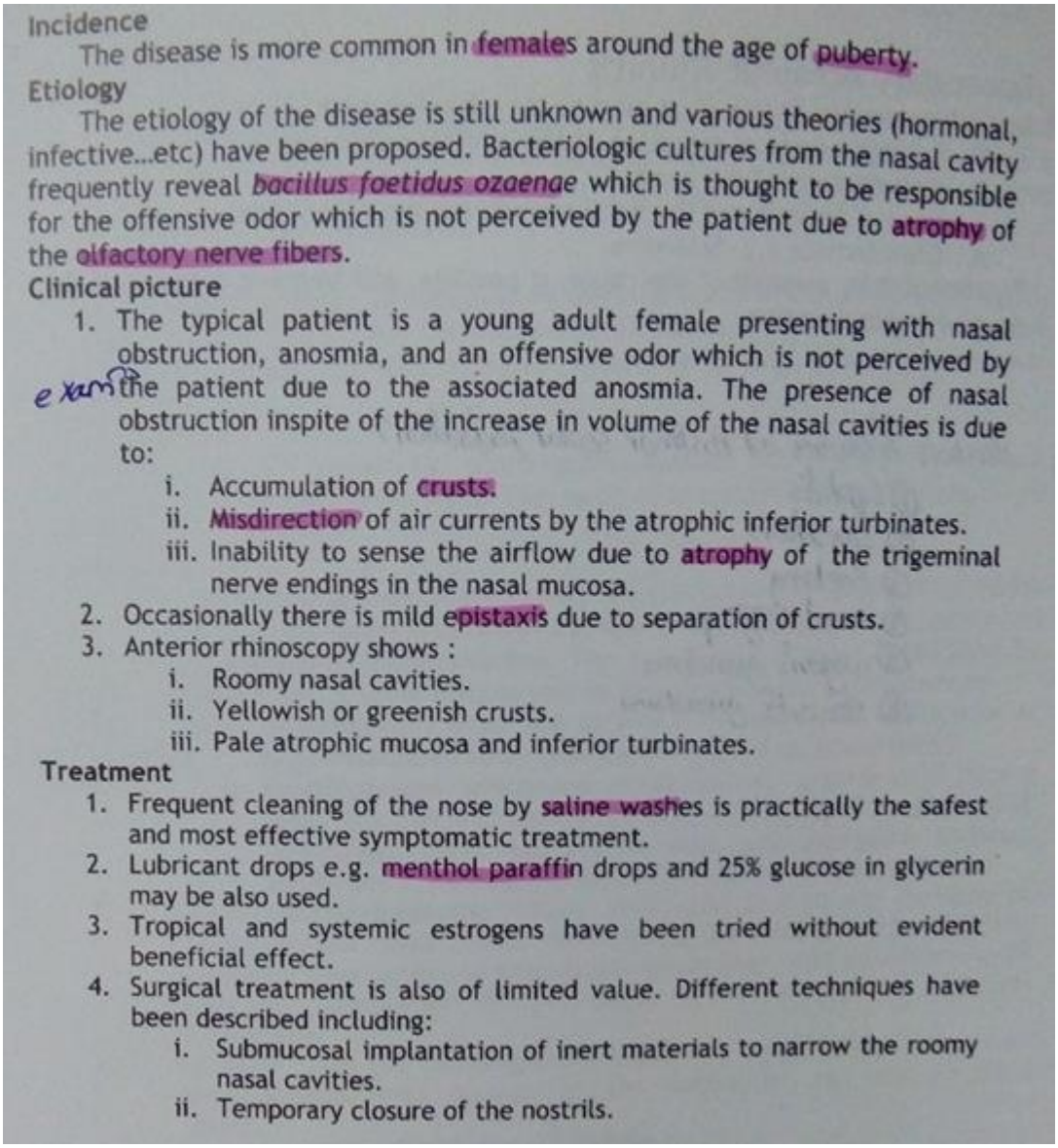
Other measures: In some cases the above measure may not be enough to permanently control the bleeding. In these cases other measure may be used such as:

- Endoscopic cautery of the sphenopalatine artery.
- Clipping of the maxillary artery through the posterior wall of the maxillary sinus (trans-antral approach).
- Clipping of the anterior ethmoidal artery as they penetrate the medial wall of the orbit.

6. Discuss primary atrophic rhinitis.

Definition

A clinical condition characterized by atrophy of the nasal mucosa, increased nasal patency, offensive odor, crust formation and anosmia



Incidence

The disease is more common in females around the age of puberty.

Etiology

The etiology of the disease is still unknown and various theories (hormonal, infective...etc) have been proposed. Bacteriologic cultures from the nasal cavity frequently reveal *bacillus foetidus ozaenae* which is thought to be responsible for the offensive odor which is not perceived by the patient due to atrophy of the olfactory nerve fibers.

Clinical picture

1. The typical patient is a young adult female presenting with nasal obstruction, anosmia, and an offensive odor which is not perceived by the patient due to the associated anosmia. The presence of nasal obstruction inspite of the increase in volume of the nasal cavities is due to:
    i. Accumulation of crusts.
    ii. Misdirection of air currents by the atrophic inferior turbinates.
    iii. Inability to sense the airflow due to atrophy of the trigeminal nerve endings in the nasal mucosa.
2. Occasionally there is mild epistaxis due to separation of crusts.
3. Anterior rhinoscopy shows :
    i. Roomy nasal cavities.
    ii. Yellowish or greenish crusts.
    iii. Pale atrophic mucosa and inferior turbinates.

Treatment

1. Frequent cleaning of the nose by saline washes is practically the safest and most effective symptomatic treatment.
2. Lubricant drops e.g. menthol paraffin drops and 25% glucose in glycerin may be also used.
3. Tropical and systemic estrogens have been tried without evident beneficial effect.
4. Surgical treatment is also of limited value. Different techniques have been described including:
    i. Submucosal implantation of inert materials to narrow the roomy nasal cavities.
    ii. Temporary closure of the nostrils.

7. Types, clinical picture and treatment of nasal polypi.

[illegible] is also frequently associated with nasal polyps.

Pathology

1. Most nasal polyps arise from the ethmoid air cells and the middle meatus of the nose. Occasionally they arise from the middle turbinate itself. These polyps usually appear as multiple grape-like clusters, and they are notorious for their tendency to recur after surgery.

2. A solitary polyp occasionally arises from the lining mucosa of the maxillary sinus and prolepses through the ostium of the sinus towards the choana of the nose hence the name "antrochoanal polyp".

Clinical picture

A. **Symptoms:**
The patient usually has a long history of troublesome vasomotor or less frequently, true allergic rhinitis. When polypi develops *nasal obstruction* becomes more complete and not adequately relieved by vasoconstrictor drops. Also *hyposmia* becomes more evident, and frequently the patient becomes totally *anosmic*.

B. **Signs:**
   1. The polypi usually appear as bilateral multiple pale glistening soft gelatinous masses with smooth surface (grape-like clusters). They appear initially in the middle meatus, and then fill the nasal cavity and my even protrude through the nostrils.
   2. The nasal mucosa usually appears pale and edematous.

Investigations

CT scans are important for the planning of surgical treatment if indicated.

Allergy test may be done to prove or exclude true allergic rhinitis.

Treatment

1. Treatment of underlying vasomotor or allergic rhinitis.
2. Although systemic steroids may induce evident regression or sometime disappearance of the polyps, recurrence usually occur on cessation of treatment. Therefore, *topical steroids with oral antihistamines* are the preferred drug treatment for nasal polyps. They are also indicated after surgery to decrease the incidence of postoperative recurrence.
3. The primary indication for surgery is bilateral complete nasal obstruction markedly degrading the quality of life of the patient and/or interfering with his sleep:
   a. *Simple polypectomy* may be done, even as an office procedure, just to open the nasal airway.
   b. *Endoscopic polypectomy* with ethmoidectomy has a much lower recurrence rate than simple polypectomy.

Antrochoanal Polyp

Antrochoanal polyp is a solitary polyp that originates from the maxillary antrum, and then passes through the ostium of the sinus and the posterior nasal openings (choana) to the nasopharynx.

Clinical pictures

1. The patient frequently has a long history of allergic or vasomotor rhinitis and present with their typical symptoms and signs.
2. Unilateral constant nasal obstruction.
3. Endoscopy or posterior rhinoscopy is essential to see the polyp. In some cases however the polyp is so big and can be seen easily behind the uvula.

Investigations

CT scan of the nose and paranasal sinuses which also important for planning the necessary surgery.

Treatment

1. Endoscopic removal after widening the orifice of the maxillary sinus (middle meatal antrostomy).
2. Removal through a Caldwell-Luc approach (sublabial antrostomy).

8. Enumerate indications of maxillary sinus puncture.

Indications

Diagnostic:

To confirm the presence of infection in suspected cases of sinusitis (proof puncture).

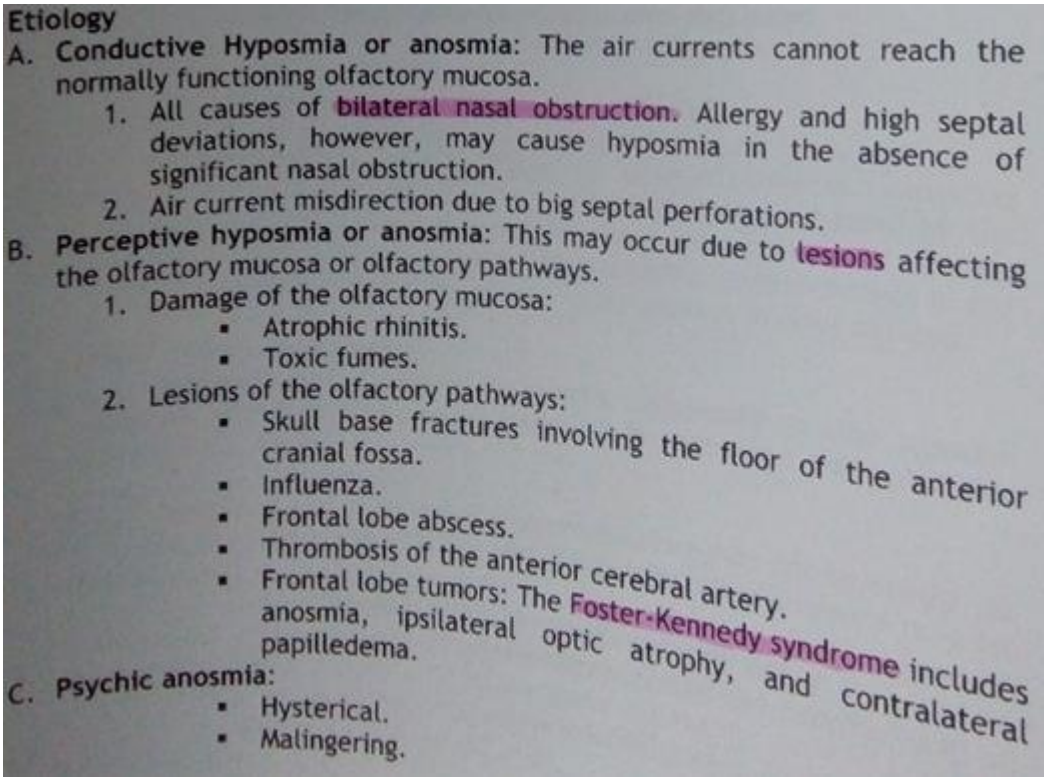
Culture and sensitivity studies of the retained sinus discharge.

Cytological examination of the fluid may help the diagnosis of early antral cancers.

Therapeutic:

- Treatment of cases of subacute and chronic maxillary sinusitis that do not respond fully to medical treatment.
- Treatment of fungal sinusitis.
- Treatment of severe Barotraumatic sinusitis.

9. Enumerate causes of hyposmia and anosmia.(3)



Etiology

A. **Conductive Hyposmia or anosmia**: The air currents cannot reach the normally functioning olfactory mucosa.
   1. All causes of bilateral nasal obstruction. Allergy and high septal deviations, however, may cause hyposmia in the absence of significant nasal obstruction.
   2. Air current misdirection due to big septal perforations.

B. **Perceptive hyposmia or anosmia**: This may occur due to lesions affecting the olfactory mucosa or olfactory pathways.
   1. Damage of the olfactory mucosa:
      - Atrophic rhinitis.
      - Toxic fumes.
   2. Lesions of the olfactory pathways:
      - Skull base fractures involving the floor of the anterior cranial fossa.
      - Influenza.
      - Frontal lobe abscess.
      - Thrombosis of the anterior cerebral artery.
      - Frontal lobe tumors: The Foster-Kennedy syndrome includes anosmia, ipsilateral optic atrophy, and contralateral papilledema.

C. **Psychic anosmia**:
   - Hysterical.
   - Malingering.

10. Diagnosis and treatment of allergic rhinitis.

Clinical Picture

A. Symptoms:
   1. Sneezing especially in the morning.
   2. Bilateral watery nasal discharge.
   3. Nasal obstruction.
   4. Hyposmia even in the absence of nasal obstruction.
   5. Itching in throat.
   6. Irritant cough.
   7. Itching and watering of the eyes are common in seasonal allergic rhinitis

B. Signs:
   1. Pale bluish, moist, edematous nasal mucosa.
   2. Swollen inferior turbinates.
   3. Occasionally there are nasal polypi.
   4. Puffiness and blue circles around the eyes.

5. A skin crease may be evident immediately above the nasal tip due to frequent rubbing of the nose (allergy salute).

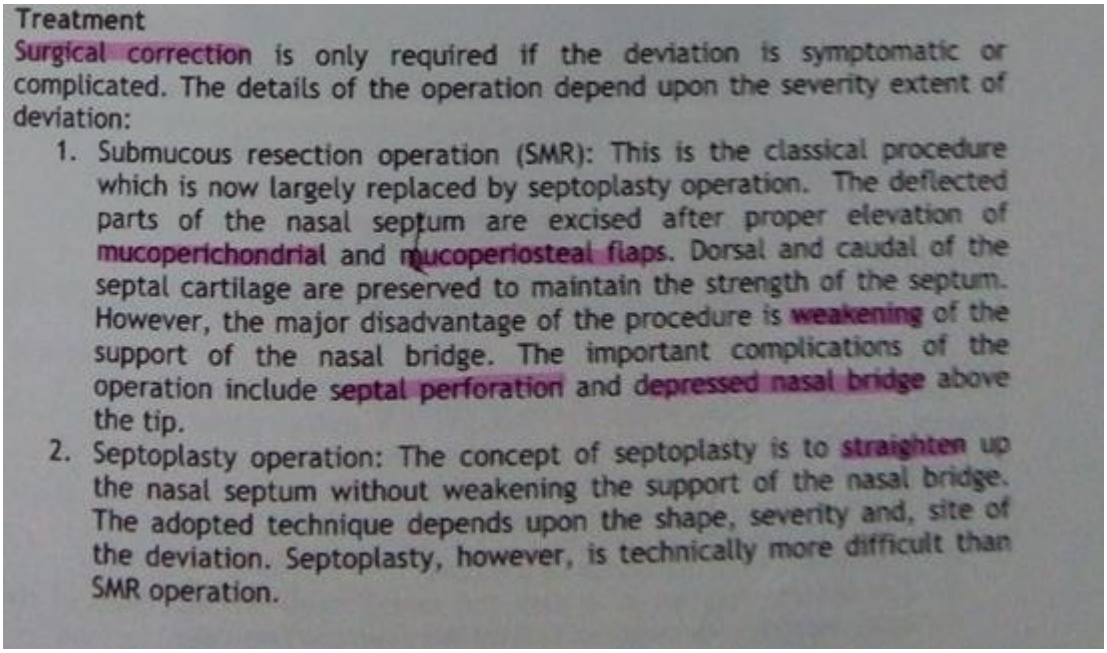
Diagnosis

1. Examination of nasal smears for eosinophilia: This is a non specific test as eosinophils may be present in the nasal discharge or some types of non-allergic rhinitis.
2. Skin tests (prick or intradermal) not specific exam
3. RAST (Radioallergosorbent test) test to measure circulating IgE antibodies for specific antigens. It more sensitive but much more expensive than skin tests.

Treatment

1. Avoidance and/or elimination of the offending allergens whenever possible.
2. Drug treatment:
   i. Topical steroid sprays are frequently very effective in controlling the allergic symptoms. They have few side effects on long term use and their systemic absorption is minimal.
   ii. Oral antihistamines selectively block H1 receptors and minimize the effect of the released histamine. The newer non-sedating, long-acting antihistamines allowed more comfortable dosing schedules and avoided the sleepiness typically induced by older preparations. Antihistamines are occasionally mixed with decongestant preparations.
   iii. Topical antihistamines are now available but they are less effective than topical steroids.
   iv. Topical sodium cromoglycate which is a mast cell stabilizing drug.
   v. Oral steroids are the most potent and least safe anti-allergic drug. They may be prescribed for severe cases in short tapering courses taking in considerations the well known contraindications such as diabetes and hypertension.
3. Hyposensitization therapy: This involves injection of the accused allergens in gradually rising concentrations in order to encourage the formation of blocking IgG antibodies that will prevent the allergens from attaching to mast cells. The results of this therapy, however, are unpredictable and its success rates are generally low (around 30-40%).
4. Surgery has a limited role in allergic patients and is better to be avoided unless it is absolutely indicated, e.g. to relieve gross intolerable nasal obstruction or to open significantly obstructed drainage of the sinuses.

11. Discuss antrochoanal polyp.
12. Enumerate stages of rhinoscleroma.
    - Catarrhal stage
    - Hypertrophic stage: over growth of dense fibrous tissue.
    - Atrophic stage: reabsorption and breakdown of tissue
    - Cicatricial stage: scar left by the formation of new connective tissue over-healing wound
13. Treatment of deviated nasal septum.



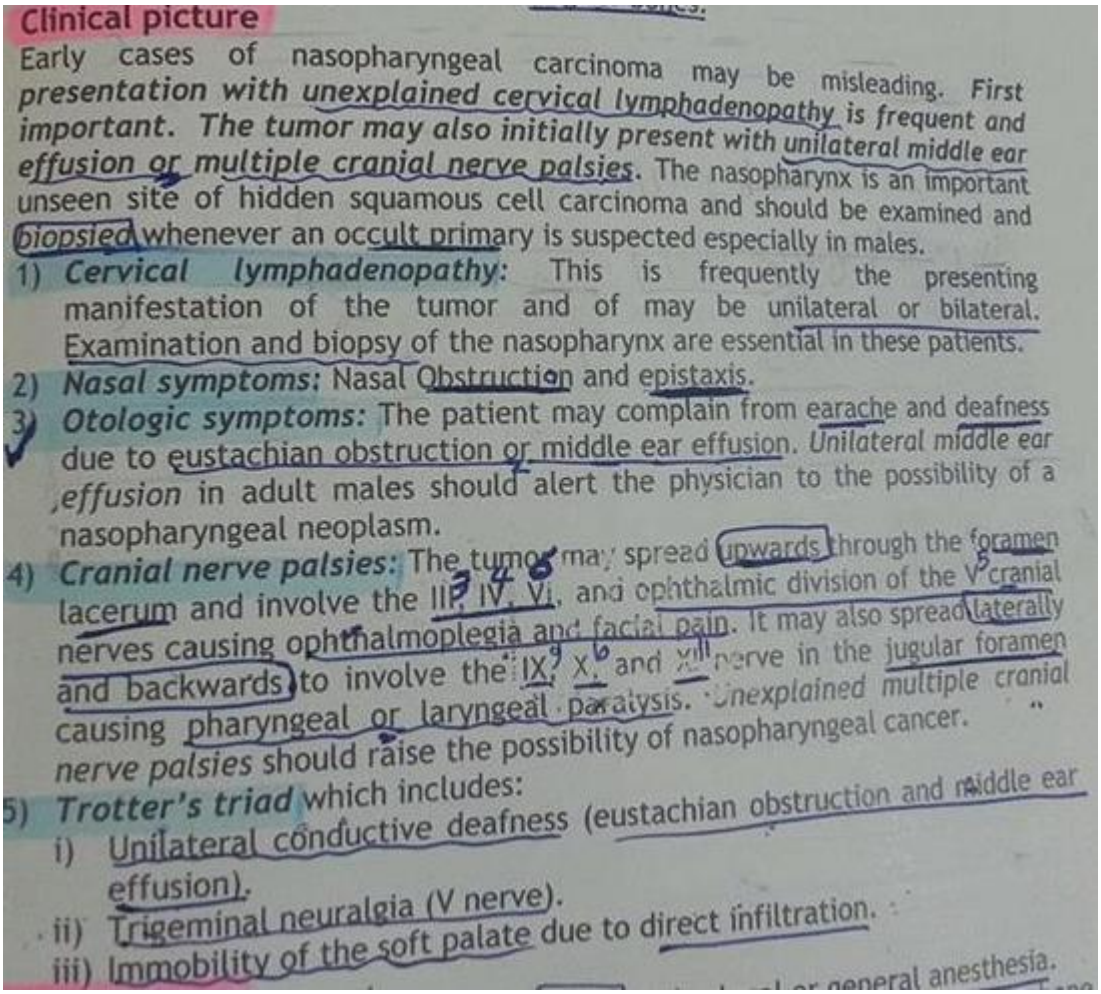
Treatment

Surgical correction is only required if the deviation is symptomatic or complicated. The details of the operation depend upon the severity extent of deviation:

1. Submucous resection operation (SMR): This is the classical procedure which is now largely replaced by septoplasty operation. The deflected parts of the nasal septum are excised after proper elevation of mucoperichondrial and mucoperiosteal flaps. Dorsal and caudal of the septal cartilage are preserved to maintain the strength of the septum. However, the major disadvantage of the procedure is weakening of the support of the nasal bridge. The important complications of the operation include septal perforation and depressed nasal bridge above the tip.
2. Septoplasty operation: The concept of septoplasty is to straighten up the nasal septum without weakening the support of the nasal bridge. The adopted technique depends upon the shape, severity and, site of the deviation. Septoplasty, however, is technically more difficult than SMR operation.

1. PHARYNX&LARYNX

Clinical picture of nasopharyngeal carcinoma.



Clinical picture

Early cases of nasopharyngeal carcinoma may be misleading. ***First presentation with unexplained cervical lymphadenopathy is frequent and important. The tumor may also initially present with unilateral middle ear effusion or multiple cranial nerve palsies***. The nasopharynx is an important unseen site of hidden squamous cell carcinoma and should be examined and biopsied whenever an occult primary is suspected especially in males.

1) ***Cervical lymphadenopathy:*** This is frequently the presenting manifestation of the tumor and of may be unilateral or bilateral. Examination and biopsy of the nasopharynx are essential in these patients.
2) ***Nasal symptoms:*** Nasal Obstruction and epistaxis.
3) ***Otologic symptoms:*** The patient may complain from earache and deafness due to eustachian obstruction or middle ear effusion. *Unilateral middle ear effusion* in adult males should alert the physician to the possibility of a nasopharyngeal neoplasm.
4) ***Cranial nerve palsies:*** The tumor may spread upwards through the foramen lacerum and involve the III, IV, VI, and ophthalmic division of the V cranial nerves causing ophthalmoplegia and facial pain. It may also spread laterally and backwards to involve the IX, X, and XII nerve in the jugular foramen causing pharyngeal or laryngeal paralysis. *Unexplained multiple cranial nerve palsies* should raise the possibility of nasopharyngeal cancer.
5) ***Trotter's triad*** which includes:
   i) Unilateral conductive deafness (eustachian obstruction and middle ear effusion).
   ii) Trigeminal neuralgia (V nerve).
   iii) Immobility of the soft palate due to direct infiltration.

2. Predisposing factors pathology clinical picture and treatment of vocalcord carcinoma.(3)

Contraindications to partial laryngectomies:

- Fixed cords (except Supracricoid laryngectomies)
- Cartilage invasion
- Subglottic extension
- If tumor extends 5mm from the foramen caecum in the tongue base
- Interarytenoid involvement
- Tumor spread into neck

Regional control (Neck Dissection)

1. **Prophylactic (Elective) in N0:**
2. **Therapeutic N1-3:**
   a. **Selective**: i
   b. **Modified RND :** radical excision of the neck removing the whole neck except the carotid artery and prevertebral muscles and cervical plexus with preservation of either:
      I. **Accessory N**
      II. **Jugular V**
      III. **a +b + SCM**
   c. **Radical Neck**
   d. **Extended:** To the occipital triangle or superior mediastinum

Distant Control

This indicated in distant metastasis and chemotherapy and radiotherapy are both indicated. IT IS VERY RARELY SUCCESSFUL

Rehabilitation

1. **Voice**
   - **Prosthesis:** One-way valve allows air to enter esophagus through a **Tracheoesophageal Puncture** to allow esophageal speech which is produced by vibration of the pharyngoesophageal mucosa Best is Provox II inserted in the posterior wall of upper tracheal stump
   - **Esophageal speech training:** Esophageal speech is produced by vibration of pharyngoesophageal mucosa; requires trapping air in mouth, injecting air into the esophagus, and expulsion of air to create voice
   - **Electrolarynx:** Artificial Larynx: Vibrating device which produces

II. Treatment

Aim of Treatment

1. Local Control: Directed towards the tumor
2. Regional Control: Directed towards the lymph nodes
3. Distal Control: directed to distant metastasis
4. Conservation of function as much as possible without risking disease control
5. Rehabilitation: of voice and swallowing to improve the patients quality of life
6. Palliation: In advanced stage aiming at:
   a. Pain relief
   b. Maintain air way
   c. Maintain feeding

   Used in:
   a. Inoperable tumor
   b. Bad general health

- It is involved by 50-75% of laryngeal cancer
- Limited regional metastasis, primary lymphatic drainage to the prelaryngeal, pretracheal lymph nodes and rarely to levels II, III, and IV.
- vocal fold fixation suggests involvement of thyroarytenoid, lateral or posterior cricoarytenoid, and interarytenoid muscles; extension into cricoarytenoid joint; or perineural invasion

Late :(T3&T4)

1. Neck
   - Lump: lymph node
   - Fullness Thyrohyoid membrane
   - Broadening laryngeal Box
   - Tender laryngeal Box: due perichondritis

2. Cord Fixation. Causes:
   - Mechanical: weight of the tumor preventing cord movement
   - Infiltrative: of thyroarytenoid muscle or cricoarytenoid joint
   - Paralytic: infiltration of the nerve fibers to the muscles(very late)

Clinical Picture

Type of patient:

- Male > 50 ys (male :female =22-25:1 in Egypt, 8-12:1 Abroad)
- Rural >> Urban (Now a days)
- Smoker & Ex-Smoker*(Carcinoma in situ cells take 10-20 ys to become invasive)
- Refluxer
- Spices Eater
- Alcohol Drinker (not common in Egypt now-a-days)

Symptoms:

Early:

1. Change of Voice
2. Hoarseness
3. F.B. Sensation
4. Otalgia
5. Irritative Cough
6. Sense of air way Obstruction

Late:

1. Hot potato vice: large supraglottic tumors
2. Stridor: biphasic(long inspiratory and short expiratory)suggestive of subglottic extension of glottic,
3. Dysphagia: in supraglottic tumors
4. Lump in the Neck: More common in supraglottic tumors presenting as lymph node
5. Pain: suggestive malignant perichondritis
6. Blood Tinged Sputum: due tumor necrosis

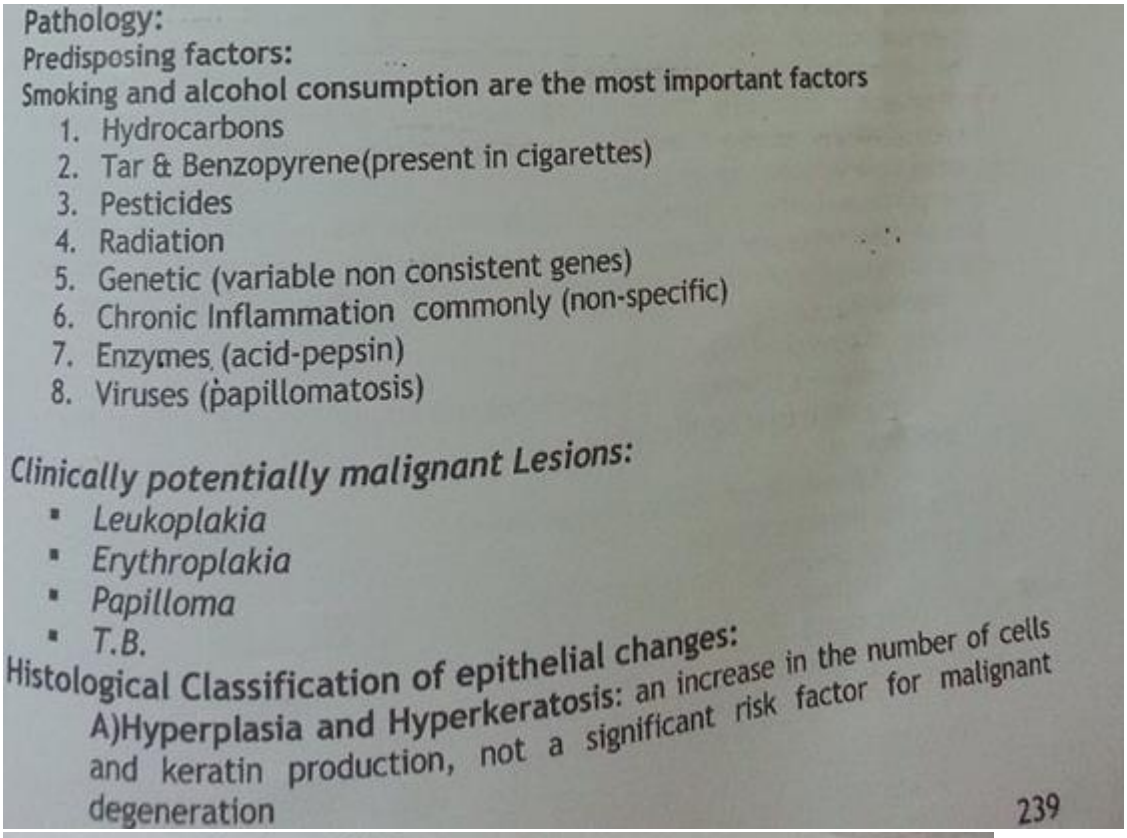
Very Late:

1. Weight Loss
2. Fetor oris: due to tumor necrosis

Signs:

Early(T1 & T2)

1. Neck Free, except Marginal Ts
2. Laryngeal Examination: Office indirect laryngoscopy (with a mirror) or Office direct laryngoscopy after surface anesthesia (xylocaine10% spray) with either a flexible nasopharyngolaryngoscope or a rigid 70 or 90 degrees laryngoscope (per oral). This will show:
   - Disturbed Vascular Pattern of the vocal cord
   - Thickening/Mass/Ulcer, according to the its location



Pathology:

Predisposing factors:

Smoking and alcohol consumption are the most important factors

1. Hydrocarbons
2. Tar & Benzopyrene(present in cigarettes)
3. Pesticides
4. Radiation
5. Genetic (variable non consistent genes)
6. Chronic Inflammation commonly (non-specific)
7. Enzymes (acid-pepsin)
8. Viruses (papillomatosis)

Clinically potentially malignant Lesions:

- *Leukoplakia*
- *Erythroplakia*
- *Papilloma*
- *T.B.*

Histological Classification of epithelial changes:

A)Hyperplasia and Hyperkeratosis: an increase in the number of cells and keratin production, not a significant risk factor for malignant degeneration

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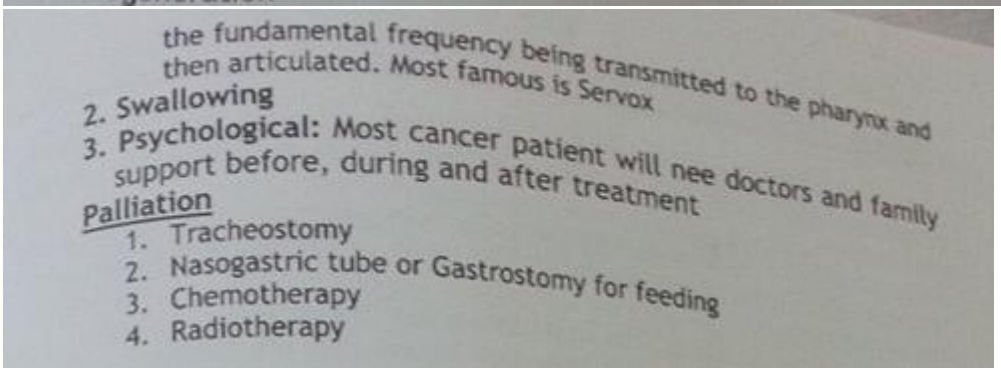


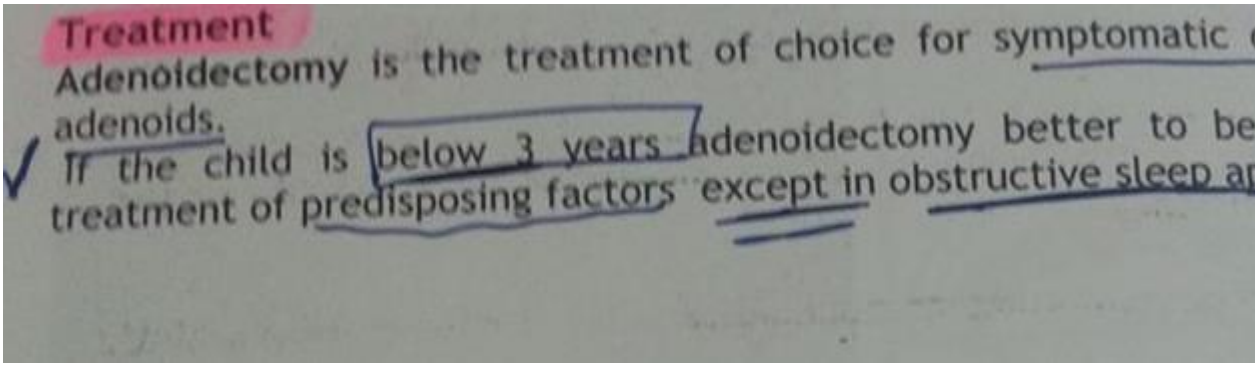
the fundamental frequency being transmitted to the pharynx and then articulated. Most famous is Servox

2. Swallowing
3. Psychological: Most cancer patient will nee doctors and family support before, during and after treatment

Palliation

1. Tracheostomy
2. Nasogastric tube or Gastrostomy for feeding
3. Chemotherapy
4. Radiotherapy

3. Clinical picture and treatment of adenoids.



Treatment

Adenoidectomy is the treatment of choice for symptomatic adenoids.

If the child is below 3 years adenoidectomy better to be treatment of predisposing factors except in obstructive sleep ap

F) Feeding problems, loss of appetite, indigestion, and vomiting due to swallowed secretions.

G) Skeletal changes including pigeon chest, Harrison's sulci, and depressed xiphisternum due to increase respiratory efforts.

H) Decreased mental performance due to sleep disturbances, hypoxia, defective hearing, and recurrent respiratory infections causing long periods of absences from school.

I) Examination of the throat frequently reveals chronic tonsils. The presence of a plug of mucous (egg white like) behind the soft palate on gagging is frequently diagnostic of the condition.

Clinical picture

A) Nasal obstruction, which results in:
   1) Mouth breathing and other features of adenoid facies.
   2) Snoring.
   3) Difficulty of suckling and eating.
   4) A characteristic hyponasal tone of the voice.

Adenoid facies:

   5) Flat expressionless face due to absence of the nasolabial folds.
   6) Open mouth and dry lips.
   7) Hitched up upper lip, projecting incisors, dry gums, infected gums, and dental caries.
   8) Inactive ala nasi and the anterior nasal openings are reduced to narrow longitudinal slits.
   9) Jaws and teeth are affected and disfigured; usually teeth of the upper jaw are irregular and crowded and there is malocclusion of upper and lower jaws.
   10) High arched palate leading to loss of contact between the tongue and palate.

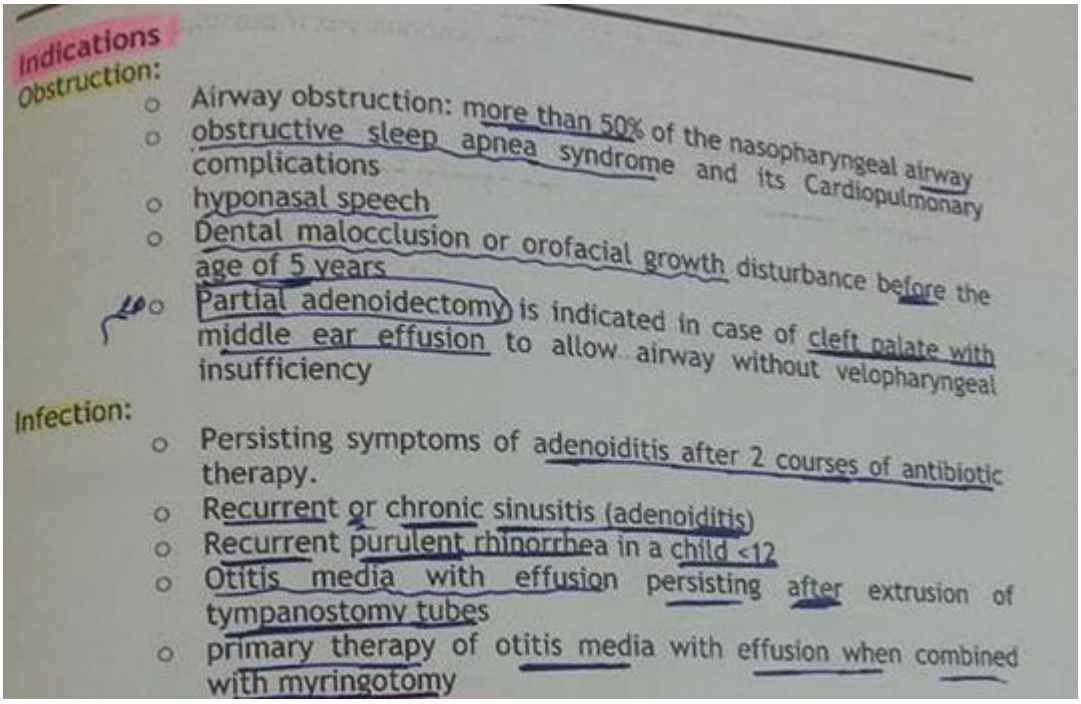
B) Mucopurulent anterior and posterior nasal discharge.

C) Recurrent ear aches and deafness due to obstruction of the eustachian tubes causing recurrent otitis media and otitis media with effusion (OME).

D) Sleep disturbances:
   1) Snoring.
   2) Sleep apnea (especially when it is associated with hypertrophic tonsils).
   3) Nightmares and nocturnal enuresis due to hypercapnia.

E) Respiratory manifestations:
   1) Irritant cough and may be laryngismus stridulus which is laryngeal spasms due to increased Co2 levels as well as irritation by the dripping postnasal discharge.

pharyngitis, laryngitis, and chest infections.



Indications

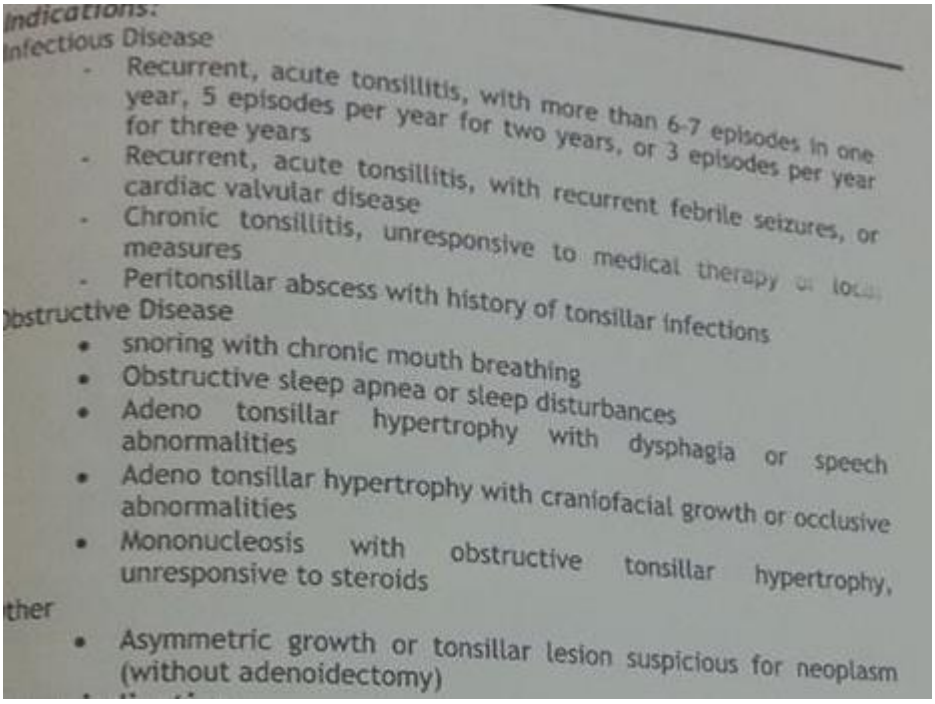
Obstruction:

- Airway obstruction: more than 50% of the nasopharyngeal airway
- obstructive sleep apnea syndrome and its Cardiopulmonary complications
- hyponasal speech
- Dental malocclusion or orofacial growth disturbance before the age of 5 years
- Partial adenoidectomy is indicated in case of cleft palate with middle ear effusion to allow airway without velopharyngeal insufficiency

Infection:

- Persisting symptoms of adenoiditis after 2 courses of antibiotic therapy.
- Recurrent or chronic sinusitis (adenoiditis)
- Recurrent purulent rhinorrhea in a child <12
- Otitis media with effusion persisting after extrusion of tympanostomy tubes
- primary therapy of otitis media with effusion when combined with myringotomy

4. Indications of tonsillectomy.



Indications:

Infectious Disease

- Recurrent, acute tonsillitis, with more than 6-7 episodes in one year, 5 episodes per year for two years, or 3 episodes per year for three years
- Recurrent, acute tonsillitis, with recurrent febrile seizures, or cardiac valvular disease
- Chronic tonsillitis, unresponsive to medical therapy or local measures
- Peritonsillar abscess with history of tonsillar infections

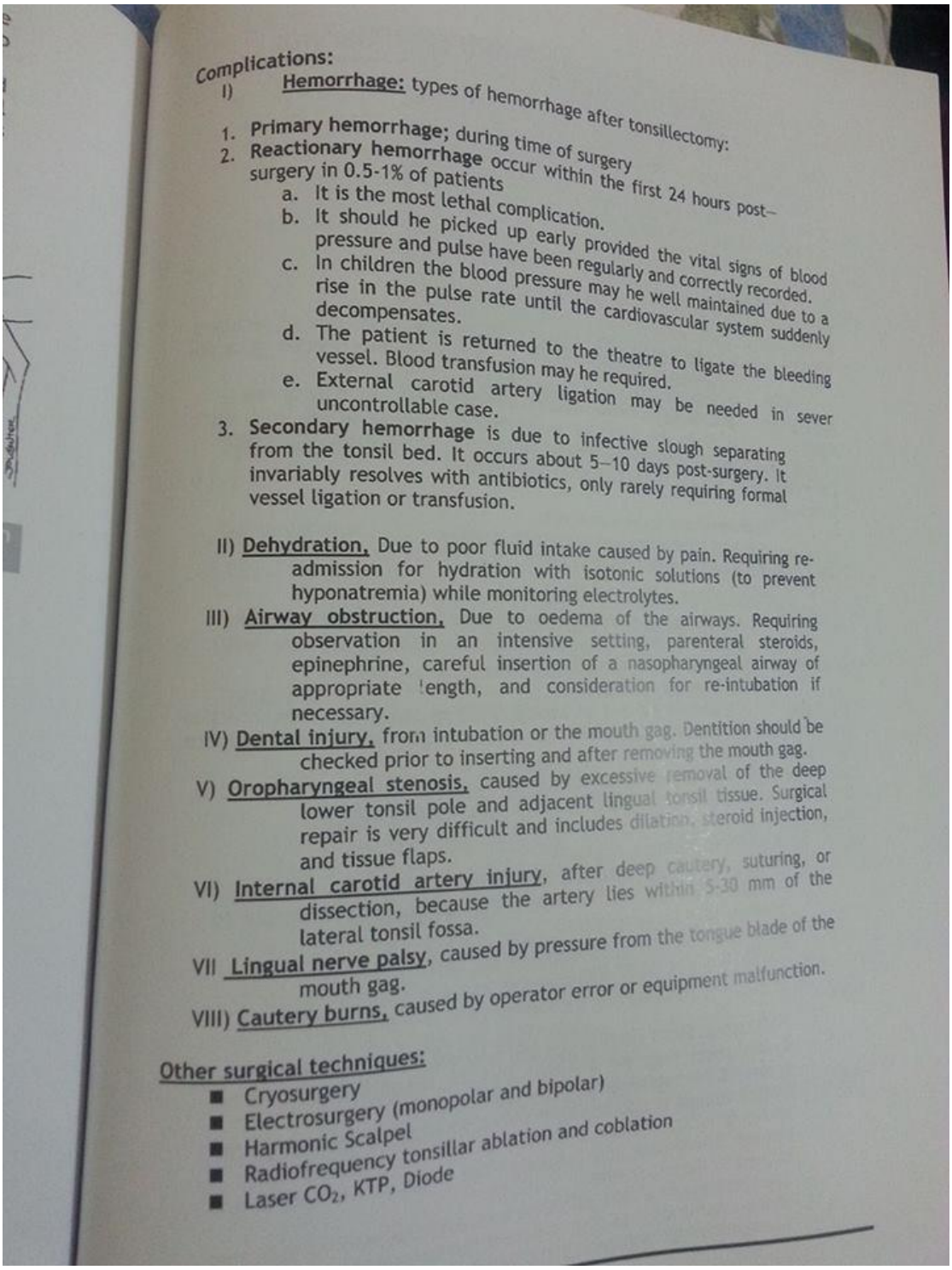
Obstructive Disease

- snoring with chronic mouth breathing
- Obstructive sleep apnea or sleep disturbances
- Adeno tonsillar hypertrophy with dysphagia or speech abnormalities
- Adeno tonsillar hypertrophy with craniofacial growth or occlusive abnormalities
- Mononucleosis with obstructive tonsillar hypertrophy, unresponsive to steroids

Other

- Asymmetric growth or tonsillar lesion suspicious for neoplasm (without adenoidectomy)

5. Complications of tonsillectomy.



Complications:

I) **Hemorrhage:** types of hemorrhage after tonsillectomy:

1. **Primary hemorrhage;** during time of surgery
2. **Reactionary hemorrhage** occur within the first 24 hours post-surgery in 0.5-1% of patients
   a. It is the most lethal complication.
   b. It should he picked up early provided the vital signs of blood pressure and pulse have been regularly and correctly recorded.
   c. In children the blood pressure may he well maintained due to a rise in the pulse rate until the cardiovascular system suddenly decompensates.
   d. The patient is returned to the theatre to ligate the bleeding vessel. Blood transfusion may he required.
   e. External carotid artery ligation may be needed in sever uncontrollable case.
3. **Secondary hemorrhage** is due to infective slough separating from the tonsil bed. It occurs about 5–10 days post-surgery. It invariably resolves with antibiotics, only rarely requiring formal vessel ligation or transfusion.

II) **Dehydration,** Due to poor fluid intake caused by pain. Requiring re-admission for hydration with isotonic solutions (to prevent hyponatremia) while monitoring electrolytes.

III) **Airway obstruction,** Due to oedema of the airways. Requiring observation in an intensive setting, parenteral steroids, epinephrine, careful insertion of a nasopharyngeal airway of appropriate length, and consideration for re-intubation if necessary.

IV) **Dental injury,** from intubation or the mouth gag. Dentition should be checked prior to inserting and after removing the mouth gag.

V) **Oropharyngeal stenosis,** caused by excessive removal of the deep lower tonsil pole and adjacent lingual tonsil tissue. Surgical repair is very difficult and includes dilation, steroid injection, and tissue flaps.

VI) **Internal carotid artery injury**, after deep cautery, suturing, or dissection, because the artery lies within 5-30 mm of the lateral tonsil fossa.

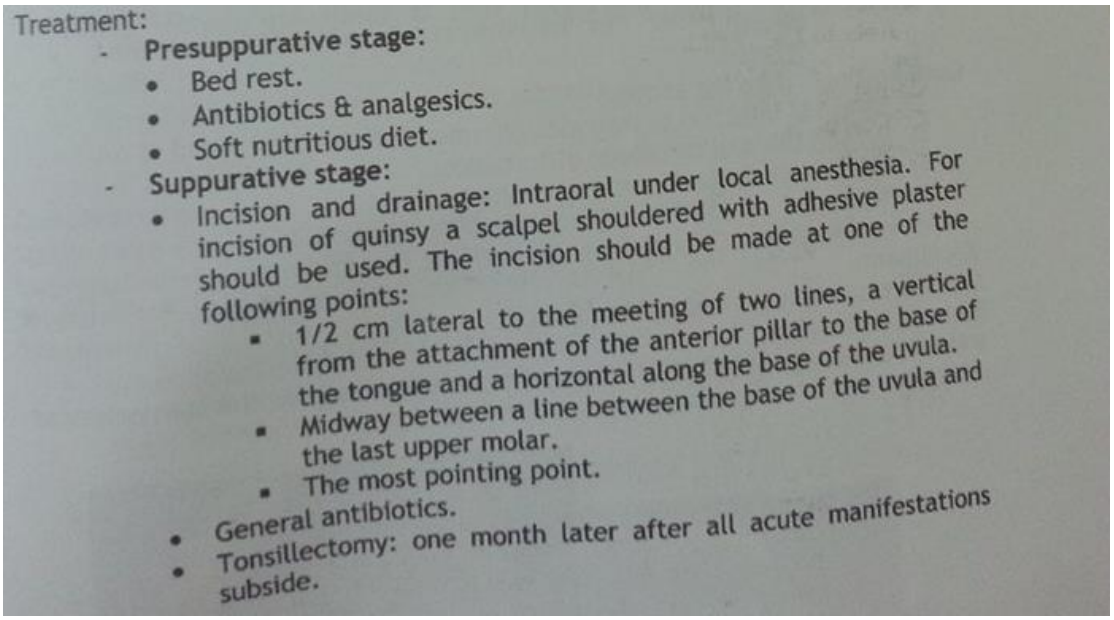
VII **Lingual nerve palsy**, caused by pressure from the tongue blade of the mouth gag.

VIII) **Cautery burns,** caused by operator error or equipment malfunction.

Other surgical techniques:

- Cryosurgery
- Electrosurgery (monopolar and bipolar)
- Harmonic Scalpel
- Radiofrequency tonsillar ablation and coblation
- Laser CO_2, KTP, Diode

6. Management of quinsy.



Treatment:

- **Presuppurative stage:**
  - Bed rest.
  - Antibiotics & analgesics.
  - Soft nutritious diet.
- **Suppurative stage:**
  - Incision and drainage: Intraoral under local anesthesia. For incision of quinsy a scalpel shouldered with adhesive plaster should be used. The incision should be made at one of the following points:
    - 1/2 cm lateral to the meeting of two lines, a vertical from the attachment of the anterior pillar to the base of the tongue and a horizontal along the base of the uvula.
    - Midway between a line between the base of the uvula and the last upper molar.
    - The most pointing point.
  - General antibiotics.
  - Tonsillectomy: one month later after all acute manifestations subside.

7. Clinical picture of bilateral vocal cord paralysis.

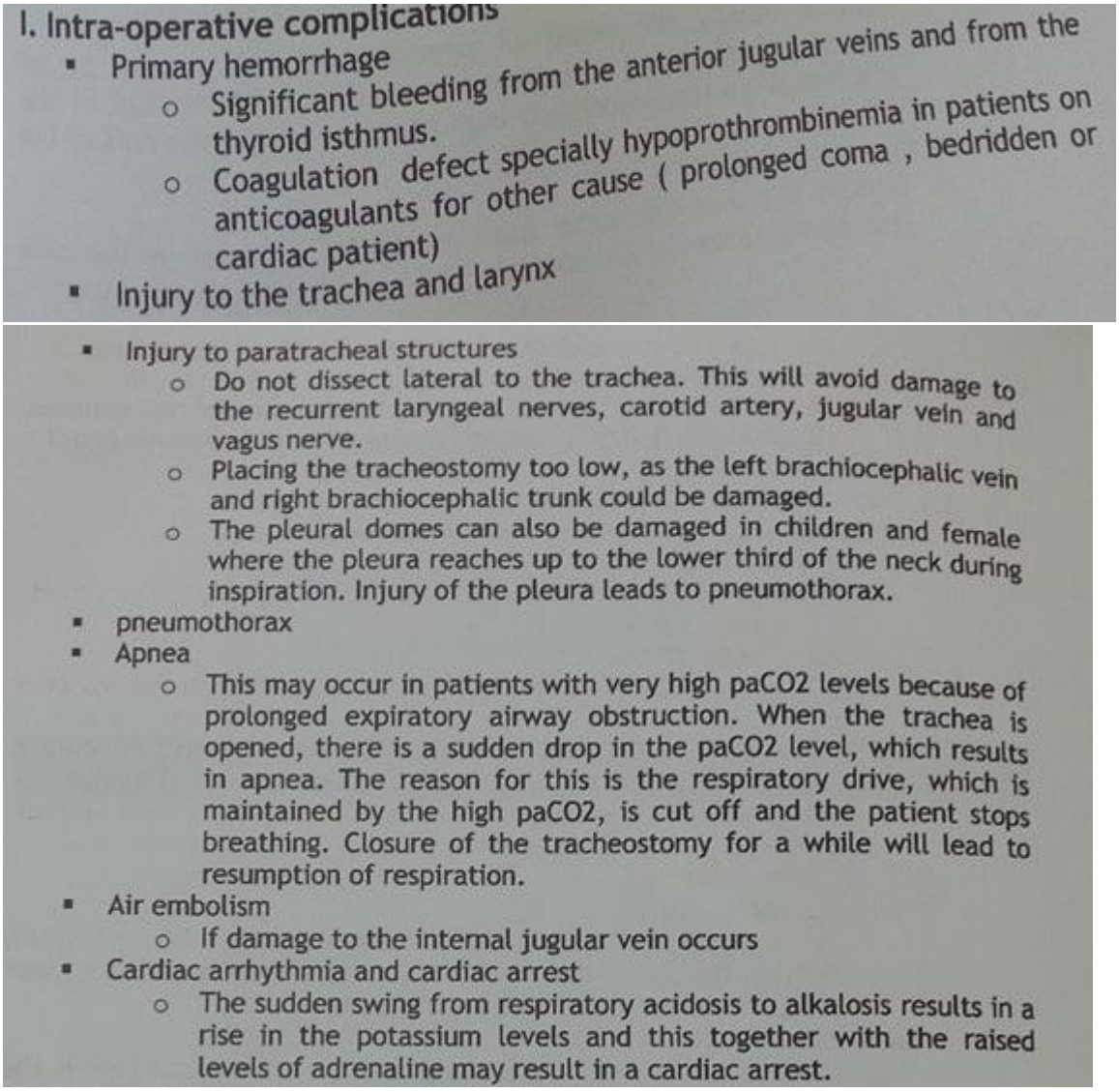
Bilateral abductor paralysis

Vocal cord are near the midline so the patient complains of stridor which increases on exertion or during upper respiratory infection. The voice is good but easily fatigues.

Bilateral adductor paralysis

Vocal cords are away from the midline and cannot close during phonation or during swallowing so the patient has aphonia and recurrent aspiration.

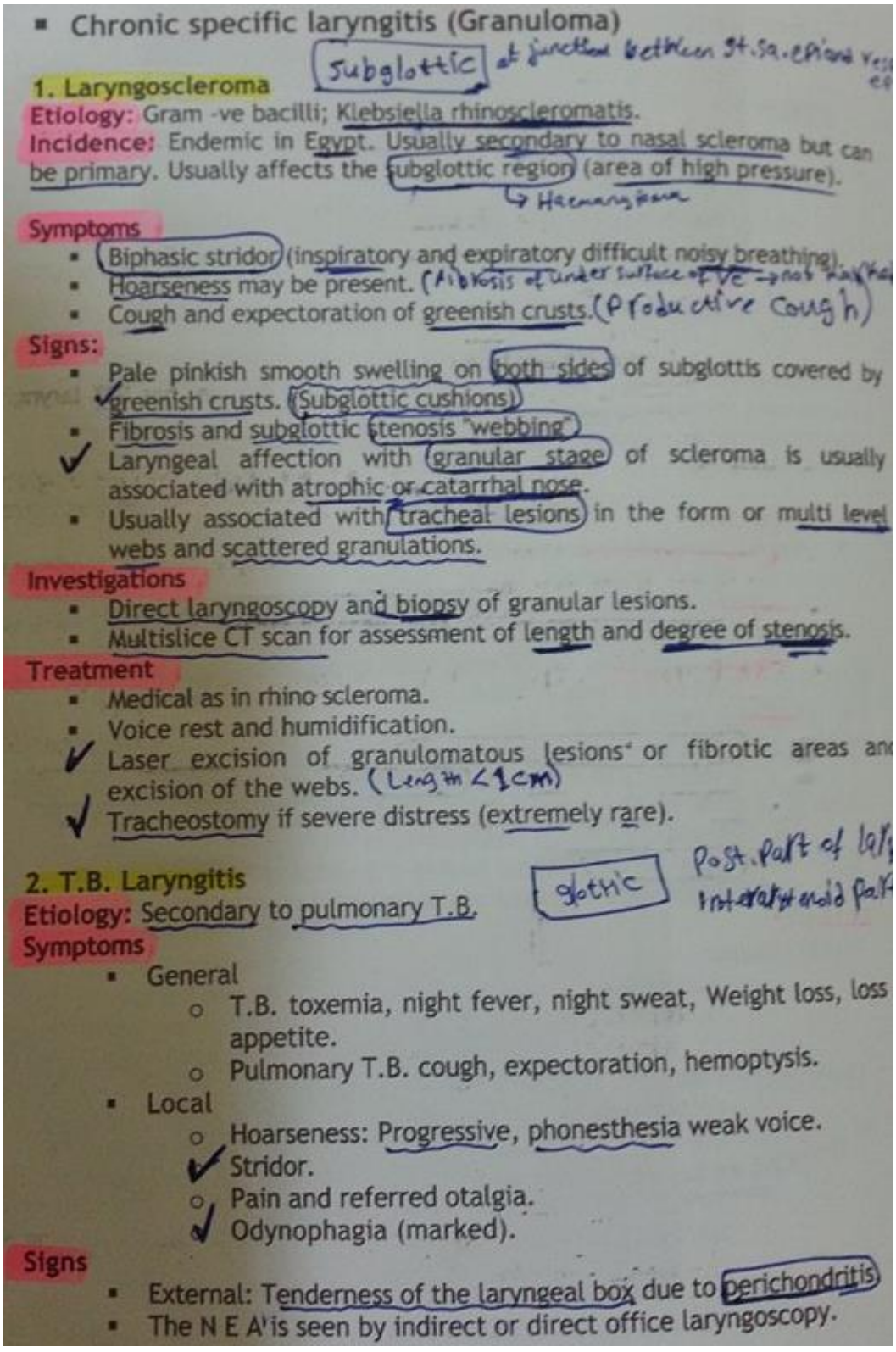
8. Operative complications of tracheostomy.



I. Intra-operative complications

- Primary hemorrhage
  - Significant bleeding from the anterior jugular veins and from the thyroid isthmus.
  - Coagulation defect specially hypoprothrombinemia in patients on anticoagulants for other cause (prolonged coma , bedridden or cardiac patient)
- Injury to the trachea and larynx
- Injury to paratracheal structures
  - Do not dissect lateral to the trachea. This will avoid damage to the recurrent laryngeal nerves, carotid artery, jugular vein and vagus nerve.
  - Placing the tracheostomy too low, as the left brachiocephalic vein and right brachiocephalic trunk could be damaged.
  - The pleural domes can also be damaged in children and female where the pleura reaches up to the lower third of the neck during inspiration. Injury of the pleura leads to pneumothorax.
- pneumothorax
- Apnea
  - This may occur in patients with very high paCO2 levels because of prolonged expiratory airway obstruction. When the trachea is opened, there is a sudden drop in the paCO2 level, which results in apnea. The reason for this is the respiratory drive, which is maintained by the high paCO2, is cut off and the patient stops breathing. Closure of the tracheostomy for a while will lead to resumption of respiration.
- Air embolism
  - If damage to the internal jugular vein occurs
- Cardiac arrhythmia and cardiac arrest
  - The sudden swing from respiratory acidosis to alkalosis results in a rise in the potassium levels and this together with the raised levels of adrenaline may result in a cardiac arrest.

9. Types of chronic specific laryngitis.



- Chronic specific laryngitis (Granuloma)

1. Laryngoscleroma

Etiology: Gram -ve bacilli; Klebsiella rhinoscleromatis.

Incidence: Endemic in Egypt. Usually secondary to nasal scleroma but can be primary. Usually affects the subglottic region (area of high pressure).

Symptoms

- Biphasic stridor (inspiratory and expiratory difficult noisy breathing).
- Hoarseness may be present.
- Cough and expectoration of greenish crusts.

Signs:

- Pale pinkish smooth swelling on both sides of subglottis covered by greenish crusts. (Subglottic cushions)
- Fibrosis and subglottic stenosis "webbing"
- Laryngeal affection with granular stage of scleroma is usually associated with atrophic or catarrhal nose.
- Usually associated with tracheal lesions in the form or multi level webs and scattered granulations.

Investigations

- Direct laryngoscopy and biopsy of granular lesions.
- Multislice CT scan for assessment of length and degree of stenosis.

Treatment

- Medical as in rhino scleroma.
- Voice rest and humidification.
- Laser excision of granulomatous lesions or fibrotic areas and excision of the webs.
- Tracheostomy if severe distress (extremely rare).

2. T.B. Laryngitis

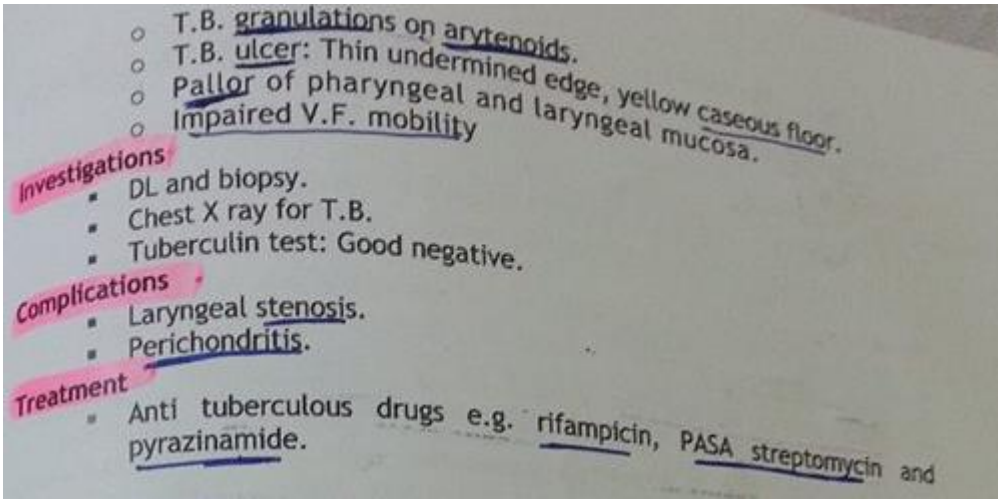
Etiology: Secondary to pulmonary T.B.

Symptoms

- General
  - T.B. toxemia, night fever, night sweat, Weight loss, loss appetite.
  - Pulmonary T.B. cough, expectoration, hemoptysis.
- Local
  - Hoarseness: Progressive, phonesthesia weak voice.
  - Stridor.
  - Pain and referred otalgia.
  - Odynophagia (marked).

Signs

- External: Tenderness of the laryngeal box due to perichondritis
- The N E A is seen by indirect or direct office laryngoscopy.



- T.B. granulations on arytenoids.
- T.B. ulcer: Thin undermined edge, yellow caseous floor.
- Pallor of pharyngeal and laryngeal mucosa.
- Impaired V.F. mobility

Investigations

- DL and biopsy.
- Chest X ray for T.B.
- Tuberculin test: Good negative.

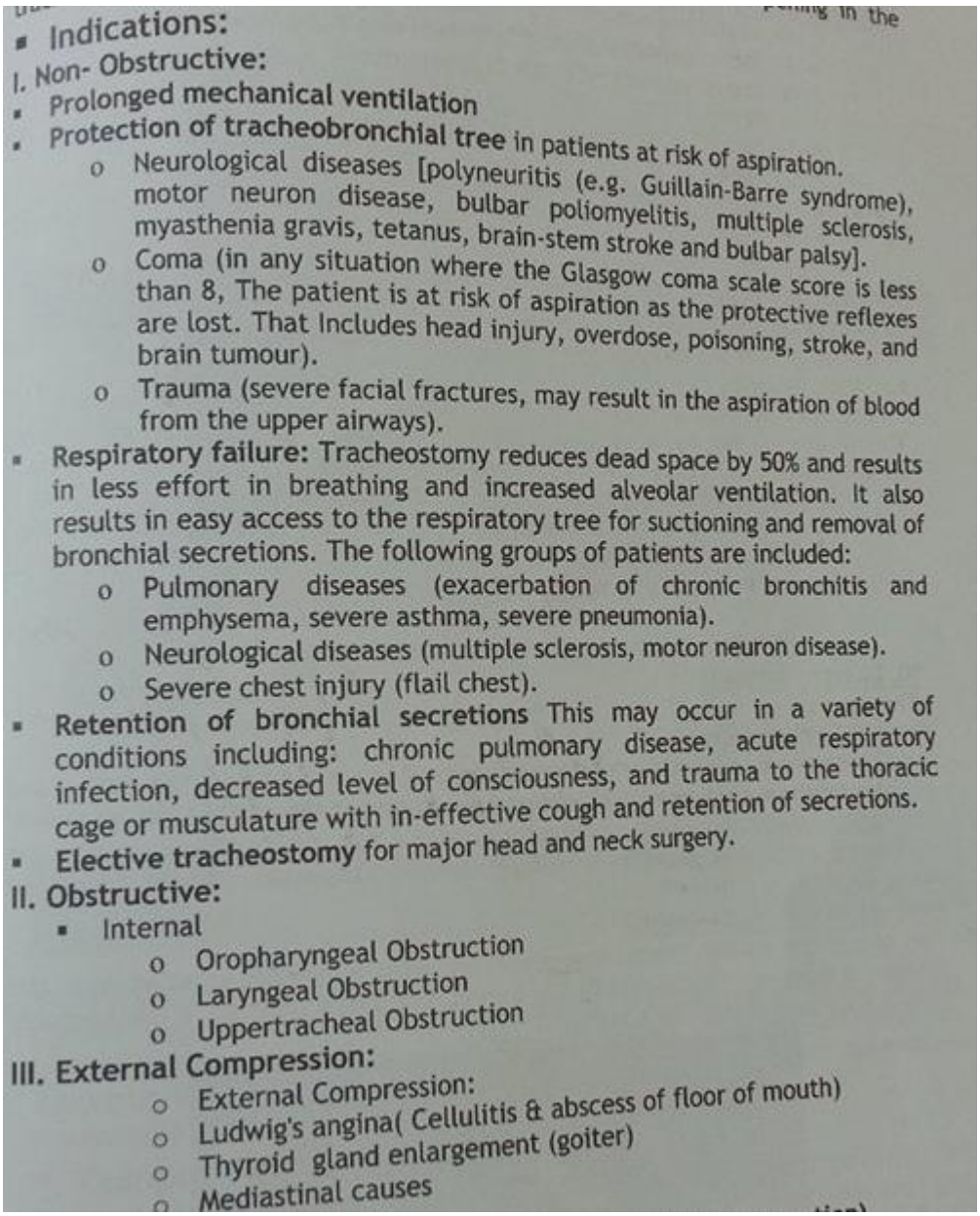
Complications

- Laryngeal stenosis.
- Perichondritis.

Treatment

- Anti tuberculous drugs e.g. rifampicin, PASA streptomycin and pyrazinamide.

10. Indications of tracheostomy.(5)



- **Indications:**

I. Non- Obstructive:

- Prolonged mechanical ventilation
- Protection of tracheobronchial tree in patients at risk of aspiration.
  - Neurological diseases [polyneuritis (e.g. Guillain-Barre syndrome), motor neuron disease, bulbar poliomyelitis, multiple sclerosis, myasthenia gravis, tetanus, brain-stem stroke and bulbar palsy].
  - Coma (in any situation where the Glasgow coma scale score is less than 8, The patient is at risk of aspiration as the protective reflexes are lost. That Includes head injury, overdose, poisoning, stroke, and brain tumour).
  - Trauma (severe facial fractures, may result in the aspiration of blood from the upper airways).
- **Respiratory failure:** Tracheostomy reduces dead space by 50% and results in less effort in breathing and increased alveolar ventilation. It also results in easy access to the respiratory tree for suctioning and removal of bronchial secretions. The following groups of patients are included:
  - Pulmonary diseases (exacerbation of chronic bronchitis and emphysema, severe asthma, severe pneumonia).
  - Neurological diseases (multiple sclerosis, motor neuron disease).
  - Severe chest injury (flail chest).
- **Retention of bronchial secretions** This may occur in a variety of conditions including: chronic pulmonary disease, acute respiratory infection, decreased level of consciousness, and trauma to the thoracic cage or musculature with in-effective cough and retention of secretions.
- **Elective tracheostomy** for major head and neck surgery.

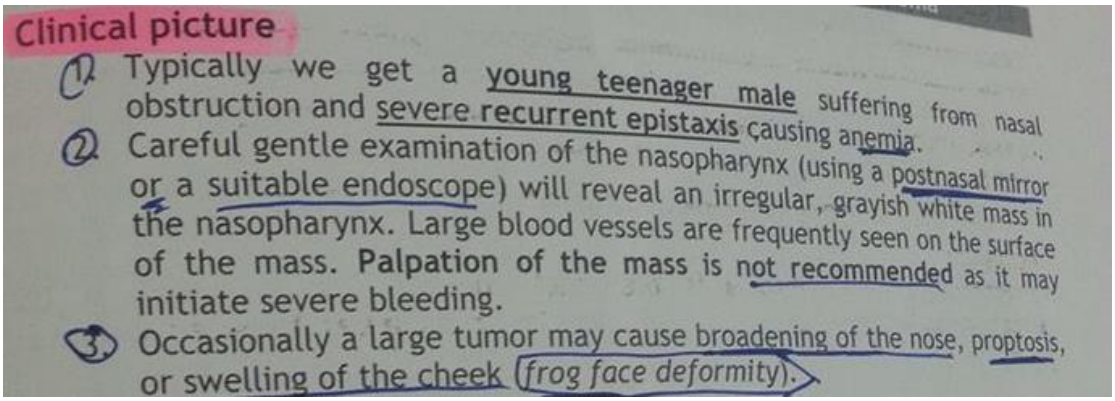
II. Obstructive:

- Internal
  - Oropharyngeal Obstruction
  - Laryngeal Obstruction
  - Uppertracheal Obstruction

III. External Compression:

- External Compression:
- Ludwig's angina(Cellulitis & abscess of floor of mouth)
- Thyroid gland enlargement (goiter)
- Mediastinal causes

11. Clinical picture and differential diagnosis of nasopharyngeal fibroma.(5)



Clinical picture

1. Typically we get a young teenager male suffering from nasal obstruction and severe recurrent epistaxis causing anemia.
2. Careful gentle examination of the nasopharynx (using a postnasal mirror or a suitable endoscope) will reveal an irregular, grayish white mass in the nasopharynx. Large blood vessels are frequently seen on the surface of the mass. Palpation of the mass is not recommended as it may initiate severe bleeding.
3. Occasionally a large tumor may cause broadening of the nose, proptosis, or swelling of the cheek (*frog face deformity*).

DDx

Adenoid

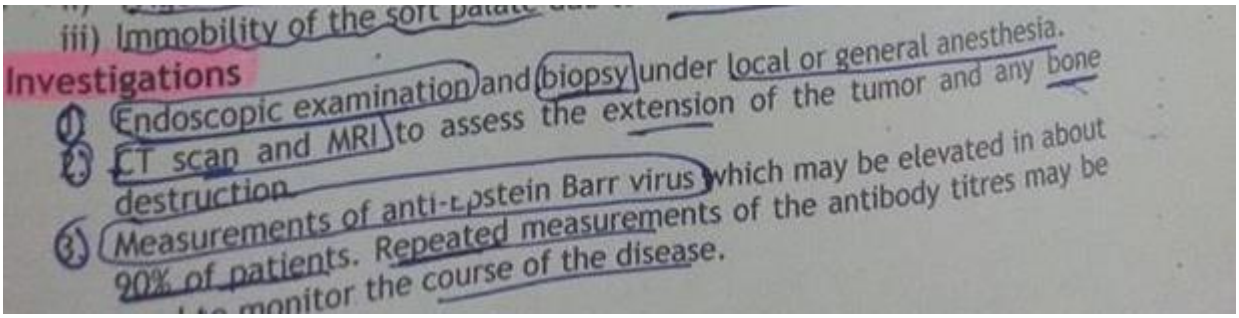
Antrochoanalpolyp

Nasopharyngeal carcinoma

Thornwaldt's cyst

Rathke's pouch cyst

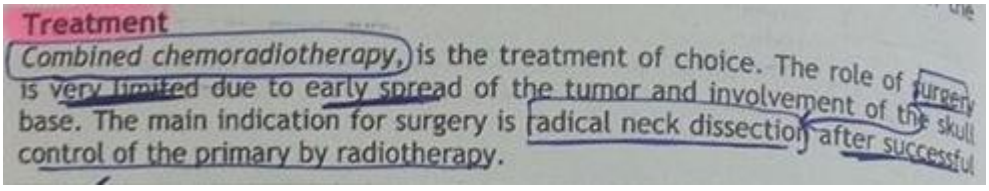
12. Diagnosis and treatment of nasopharyngeal carcinoma.



iii) Immobility of the soft palate

Investigations

1. Endoscopic examination and biopsy under local or general anesthesia.
2. CT scan and MRI to assess the extension of the tumor and any bone destruction
3. Measurements of anti-Epstein Barr virus which may be elevated in about 90% of patients. Repeated measurements of the antibody titres may be to monitor the course of the disease.

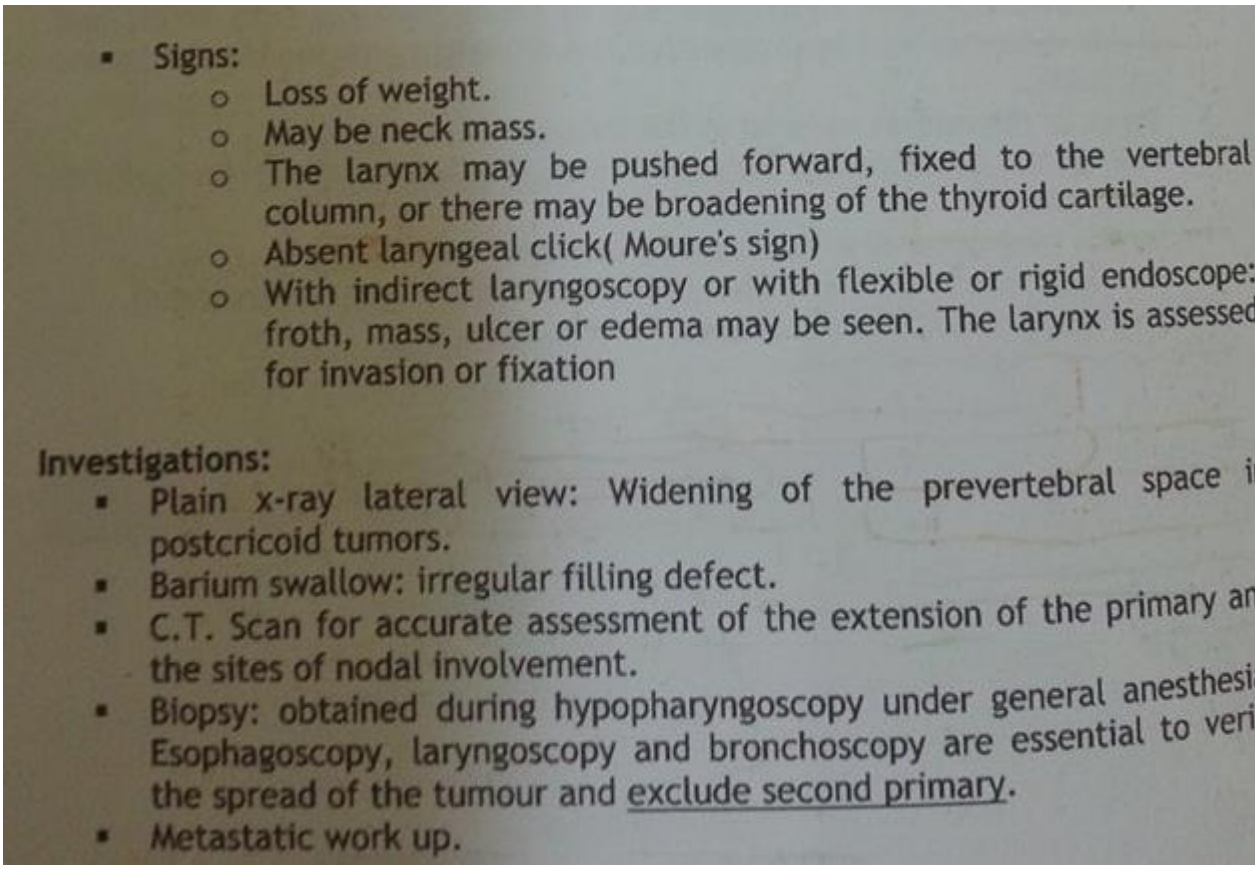


Treatment

Combined chemoradiotherapy, is the treatment of choice. The role of surgery is very limited due to early spread of the tumor and involvement of the skull base. The main indication for surgery is radical neck dissection after successful control of the primary by radiotherapy.

(+clinical picture in Q1)

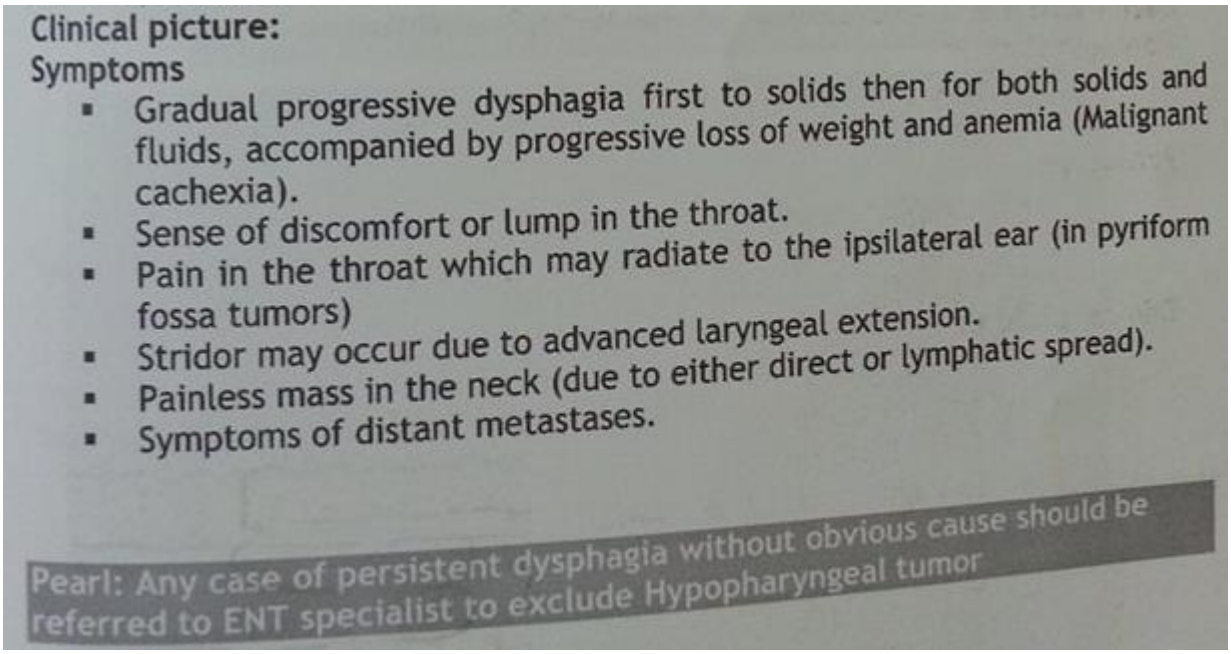
13. Diagnosis and treatment of postcricoid carcinoma.



- Signs:
  - Loss of weight.
  - May be neck mass.
  - The larynx may be pushed forward, fixed to the vertebral column, or there may be broadening of the thyroid cartilage.
  - Absent laryngeal click(Moure's sign)
  - With indirect laryngoscopy or with flexible or rigid endoscope: froth, mass, ulcer or edema may be seen. The larynx is assessed for invasion or fixation

Investigations:

- Plain x-ray lateral view: Widening of the prevertebral space i postcricoid tumors.
- Barium swallow: irregular filling defect.
- C.T. Scan for accurate assessment of the extension of the primary ar the sites of nodal involvement.
- Biopsy: obtained during hypopharyngoscopy under general anesthesi Esophagoscopy, laryngoscopy and bronchoscopy are essential to veri the spread of the tumour and exclude second primary.
- Metastatic work up.



Clinical picture:

Symptoms

- Gradual progressive dysphagia first to solids then for both solids and fluids, accompanied by progressive loss of weight and anemia (Malignant cachexia).
- Sense of discomfort or lump in the throat.
- Pain in the throat which may radiate to the ipsilateral ear (in pyriform fossa tumors)
- Stridor may occur due to advanced laryngeal extension.
- Painless mass in the neck (due to either direct or lymphatic spread).
- Symptoms of distant metastases.

Pearl: Any case of persistent dysphagia without obvious cause should be referred to ENT specialist to exclude Hypopharyngeal tumor

- **Postcricoid Region:** (40%) associated with Plummer-Vinson Syndrome, may extend into the cricoid cartilage, cricoarytenoid muscle, or cervical esophagus; 30% regional metastasis

Treatment:

- tumors of the hypopharynx may extend submucosally resulting in "skip lesions"
- Most present in advanced state with clinical cervical nodes (40-75%)

1-Treatment of the primary tumor:

Early ***Hypopharyngeal Cancer*** (T1 or T2)

- Transoral laser resection for selected cases or Partial pharyngectomy or partial pharyngolaryngectomy or total laryngectomy partial pharyngectomy with postoperative radiation Or primary radiation with surgical salvage (includes irradiating both sides of the neck),

Advanced ***Hypopharyngeal Cancer*** (T3 or T4)

- Total laryngopharyngectomy or total laryngopharyngo-esophagectomy with postoperative radiochemotherapy

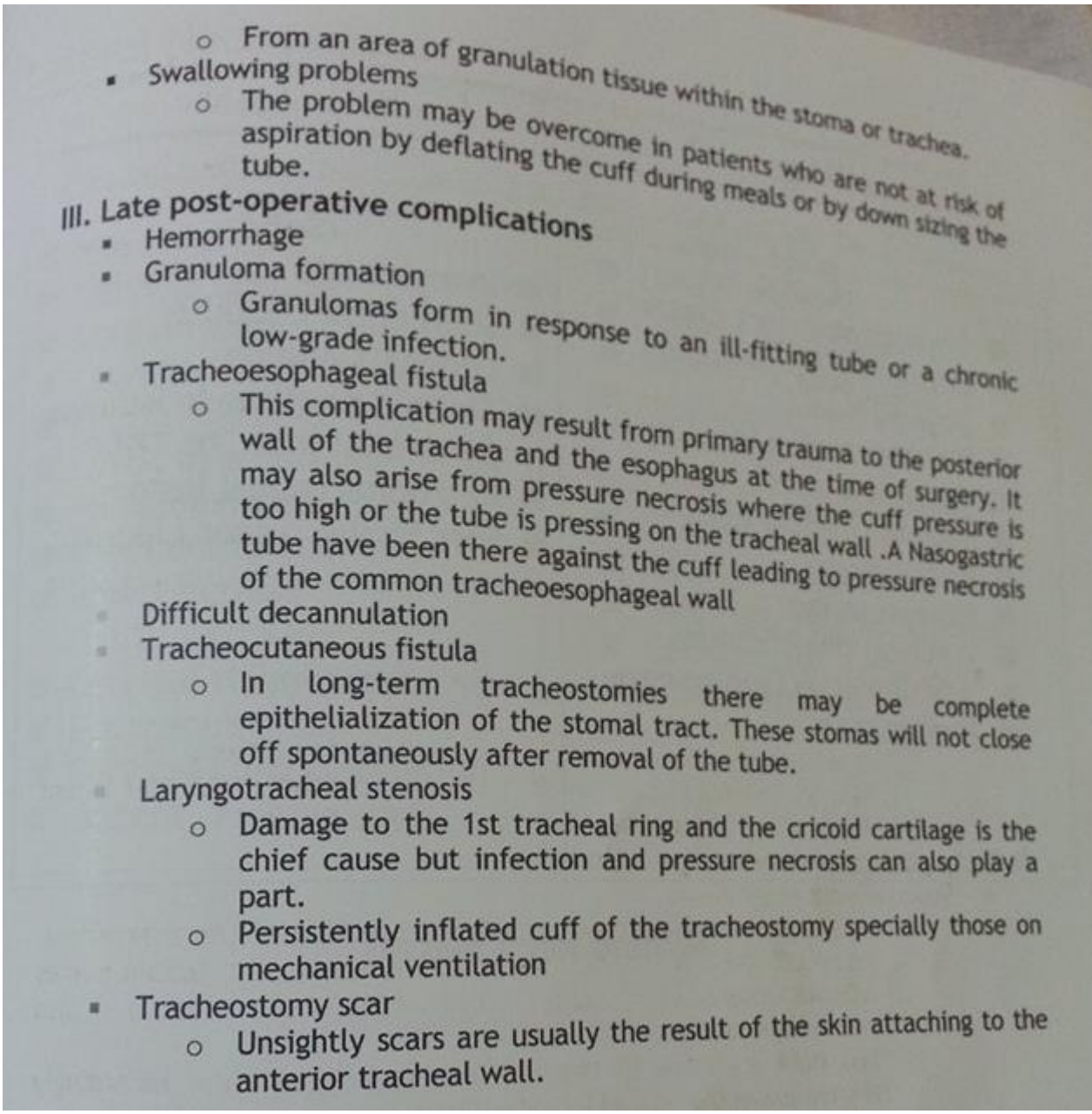
2- Treatment of the neck:

N_0: Elective ipsilateral functional neck dissection (bilateral in case of tumors that pass the midline) Or elective radiotherapy of both sides of the neck.

N_+: Ipsilateral radical neck dissection.

Pharyngeal reconstruction following tumor resection: gastric pull-up or microvascular free flap jejunal or radial forearm free flap)

14. Complications of tracheostomy.(3)



- From an area of granulation tissue within the stoma or trachea.
- Swallowing problems
  - The problem may be overcome in patients who are not at risk of aspiration by deflating the cuff during meals or by down sizing the tube.

III. **Late post-operative complications**

- Hemorrhage
- Granuloma formation
  - Granulomas form in response to an ill-fitting tube or a chronic low-grade infection.
- Tracheoesophageal fistula
  - This complication may result from primary trauma to the posterior wall of the trachea and the esophagus at the time of surgery. It may also arise from pressure necrosis where the cuff pressure is too high or the tube is pressing on the tracheal wall .A Nasogastric tube have been there against the cuff leading to pressure necrosis of the common tracheoesophageal wall
- Difficult decannulation
- Tracheocutaneous fistula
  - In long-term tracheostomies there may be complete epithelialization of the stomal tract. These stomas will not close off spontaneously after removal of the tube.
- Laryngotracheal stenosis
  - Damage to the 1st tracheal ring and the cricoid cartilage is the chief cause but infection and pressure necrosis can also play a part.
  - Persistently inflated cuff of the tracheostomy specially those on mechanical ventilation
- Tracheostomy scar
  - Unsightly scars are usually the result of the skin attaching to the anterior tracheal wall.

Complications of tracheostomy

There are many complications associated with performing a tracheostomy. Most of them can be avoided with a meticulous surgical approach and dedicated post-operative care. Emergency tracheostomy carries a 2-5 fold increase in the incidence of complications over an elective procedure.

I. Intra-operative complications

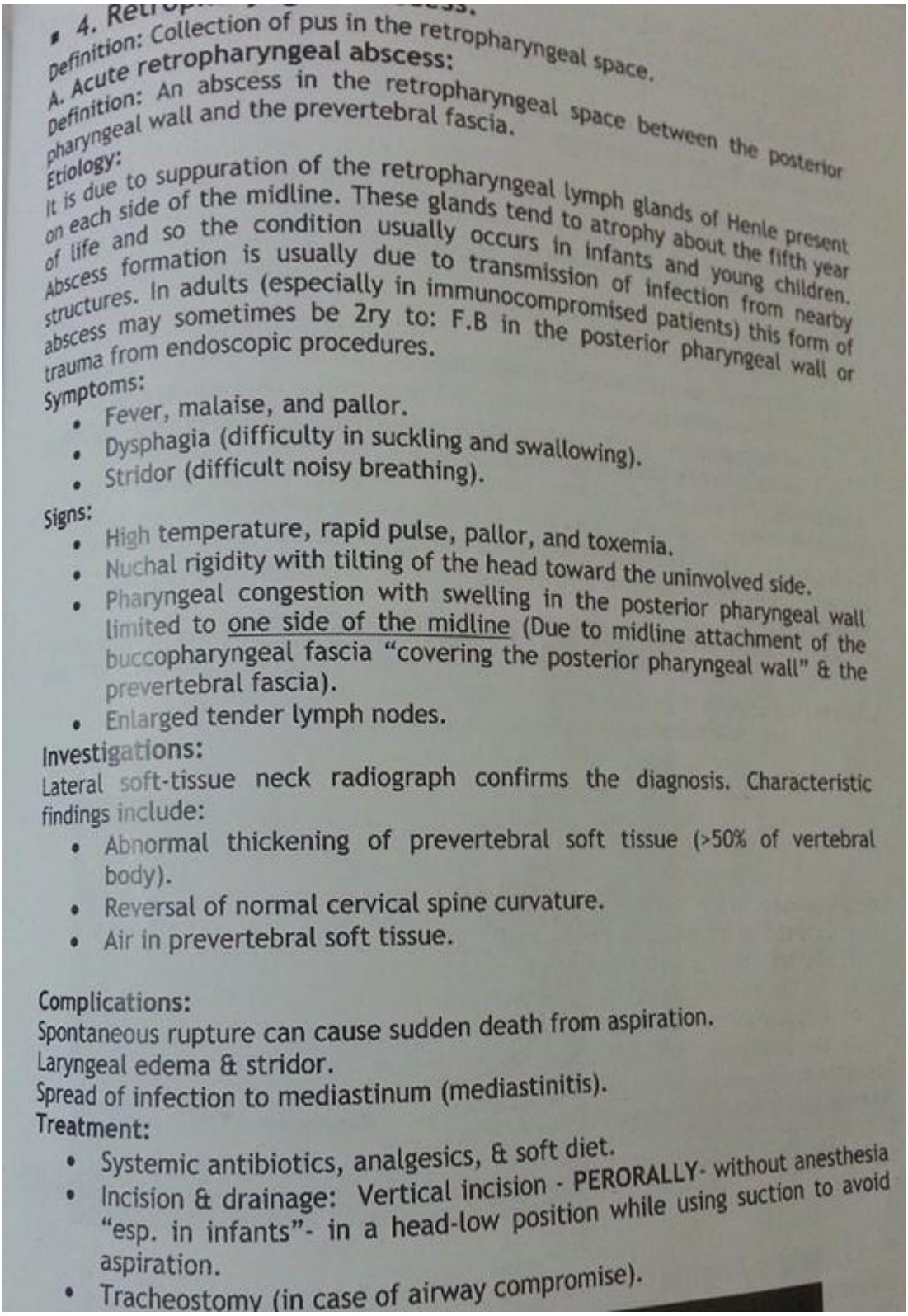
- Primary hemorrhage
  - Significant bleeding from the anterior jugular veins and from the thyroid isthmus.
  - Coagulation defect specially hypoprothrombinemia in patients on anticoagulants for other cause (prolonged coma , bedridden or cardiac patient)
- Injury to the trachea and larynx

- Injury to paratracheal structures
  - Do not dissect lateral to the trachea. This will avoid damage to the recurrent laryngeal nerves, carotid artery, jugular vein and vagus nerve.
  - Placing the tracheostomy too low, as the left brachiocephalic vein and right brachiocephalic trunk could be damaged.
  - The pleural domes can also be damaged in children and female where the pleura reaches up to the lower third of the neck during inspiration. Injury of the pleura leads to pneumothorax.
- pneumothorax
- Apnea
  - This may occur in patients with very high paCO2 levels because of prolonged expiratory airway obstruction. When the trachea is opened, there is a sudden drop in the paCO2 level, which results in apnea. The reason for this is the respiratory drive, which is maintained by the high paCO2, is cut off and the patient stops breathing. Closure of the tracheostomy for a while will lead to resumption of respiration.
- Air embolism
  - If damage to the internal jugular vein occurs
- Cardiac arrhythmia and cardiac arrest
  - The sudden swing from respiratory acidosis to alkalosis results in a rise in the potassium levels and this together with the raised levels of adrenaline may result in a cardiac arrest.

II. Early post-operative complications

- Subcutaneous emphysema
  - It is most commonly caused by non dissection of the pretracheal fascia off the trachea, leading to air escape to the fascial planes subcutaneously up and down. It can cause also pneumomediastinum.
  - Too tight a closure of the tracheostomy wound and accordingly always Leave the wound loosely closed
  - An incorrectly sized tube allowing air to escape into the wound subcutaneously instead of outwards. And thus always make sure the tube is the correct size for the patient.
- Pneumomediastinum
- Pneumothorax
  - This occurs with dissection low in the neck and damage to the pleural domes.
- Tube displacement or dislodgement
- Tube blockage/crusts
  - Avoidable by proper humidification and suctioning
- Wound infection
  - The tracheostomy wound always develops a low-grade infection with skin and respiratory tract organisms. This is usually a self-limiting infection
- Tracheal necrosis
  - This is usually the result of pressure necrosis caused by an inappropriately sized tube pressing on the posterior wall of the trachea or an inappropriately high pressure within the cuff of the tube.
- Secondary hemorrhage
  - Serious bleeding can occur from erosion of a vessel by the tube (tracheo-arterial fistula) specially the brachiocephalic artery or

15. Acute retropharyngeal abcess.



4. Retro[illegible]

Definition: Collection of pus in the retropharyngeal space.

A. Acute retropharyngeal abscess:

Definition: An abscess in the retropharyngeal space between the posterior pharyngeal wall and the prevertebral fascia.

Etiology:

It is due to suppuration of the retropharyngeal lymph glands of Henle present on each side of the midline. These glands tend to atrophy about the fifth year of life and so the condition usually occurs in infants and young children. Abscess formation is usually due to transmission of infection from nearby structures. In adults (especially in immunocompromised patients) this form of abscess may sometimes be 2ry to: F.B in the posterior pharyngeal wall or trauma from endoscopic procedures.

Symptoms:

- Fever, malaise, and pallor.
- Dysphagia (difficulty in suckling and swallowing).
- Stridor (difficult noisy breathing).

Signs:

- High temperature, rapid pulse, pallor, and toxemia.
- Nuchal rigidity with tilting of the head toward the uninvolved side.
- Pharyngeal congestion with swelling in the posterior pharyngeal wall limited to one side of the midline (Due to midline attachment of the buccopharyngeal fascia "covering the posterior pharyngeal wall" & the prevertebral fascia).
- Enlarged tender lymph nodes.

Investigations:

Lateral soft-tissue neck radiograph confirms the diagnosis. Characteristic findings include:

- Abnormal thickening of prevertebral soft tissue (>50% of vertebral body).
- Reversal of normal cervical spine curvature.
- Air in prevertebral soft tissue.

Complications:

Spontaneous rupture can cause sudden death from aspiration.

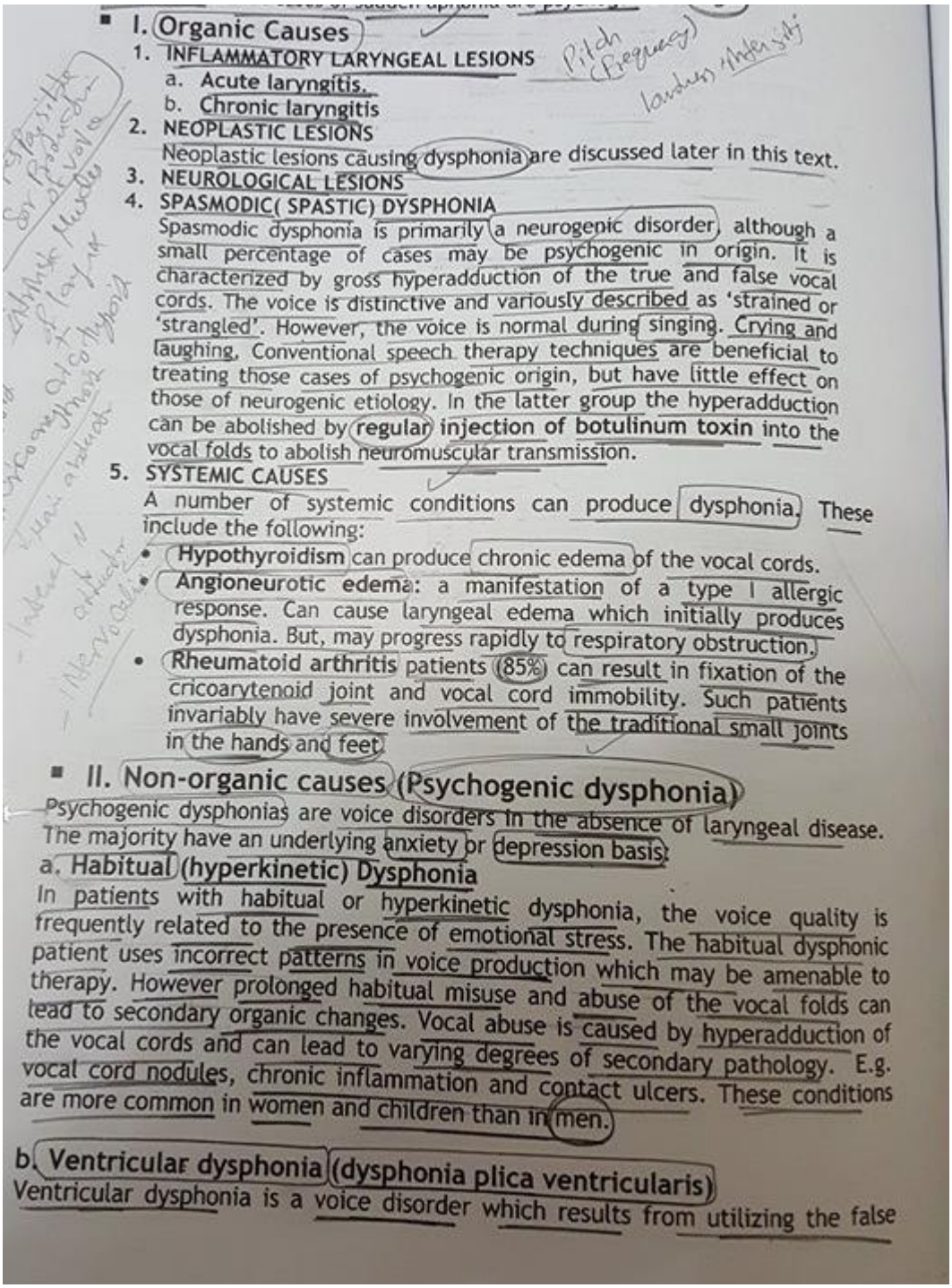
Laryngeal edema & stridor.

Spread of infection to mediastinum (mediastinitis).

Treatment:

- Systemic antibiotics, analgesics, & soft diet.
- Incision & drainage: Vertical incision - PERORALLY- without anesthesia "esp. in infants"- in a head-low position while using suction to avoid aspiration.
- Tracheostomy (in case of airway compromise).

16. Causes of hoarseness of voice.



I. Organic Causes

1. **INFLAMMATORY LARYNGEAL LESIONS**
   a. **Acute laryngitis.**
   b. **Chronic laryngitis**
2. **NEOPLASTIC LESIONS**
   Neoplastic lesions causing dysphonia are discussed later in this text.
3. **NEUROLOGICAL LESIONS**
4. **SPASMODIC(SPASTIC) DYSPHONIA**
   Spasmodic dysphonia is primarily a neurogenic disorder, although a small percentage of cases may be psychogenic in origin. It is characterized by gross hyperadduction of the true and false vocal cords. The voice is distinctive and variously described as 'strained or 'strangled'. However, the voice is normal during singing. Crying and laughing. Conventional speech therapy techniques are beneficial to treating those cases of psychogenic origin, but have little effect on those of neurogenic etiology. In the latter group the hyperadduction can be abolished by **regular injection of botulinum toxin** into the vocal folds to abolish neuromuscular transmission.
5. **SYSTEMIC CAUSES**
   A number of systemic conditions can produce dysphonia. These include the following:
   - **Hypothyroidism** can produce chronic edema of the vocal cords.
   - **Angioneurotic edema**: a manifestation of a type I allergic response. Can cause laryngeal edema which initially produces dysphonia. But, may progress rapidly to respiratory obstruction.
   - **Rheumatoid arthritis** patients (85%) can result in fixation of the cricoarytenoid joint and vocal cord immobility. Such patients invariably have severe involvement of the traditional small joints in the hands and feet.

II. Non-organic causes (Psychogenic dysphonia)

Psychogenic dysphonias are voice disorders in the absence of laryngeal disease. The majority have an underlying anxiety or depression basis.

a. Habitual (hyperkinetic) Dysphonia

In patients with habitual or hyperkinetic dysphonia, the voice quality is frequently related to the presence of emotional stress. The habitual dysphonic patient uses incorrect patterns in voice production which may be amenable to therapy. However prolonged habitual misuse and abuse of the vocal folds can lead to secondary organic changes. Vocal abuse is caused by hyperadduction of the vocal cords and can lead to varying degrees of secondary pathology. E.g. vocal cord nodules, chronic inflammation and contact ulcers. These conditions are more common in women and children than in men.

b. Ventricular dysphonia (dysphonia plica ventricularis)

Ventricular dysphonia is a voice disorder which results from utilizing the false

cords to produce vibration. It is usually seen in tense
patients with lesions of the true cords may compensate
false cords. The voice is harsh and frequently low pitch
being strangled. Examination shows the ventricular folds
during phonation.

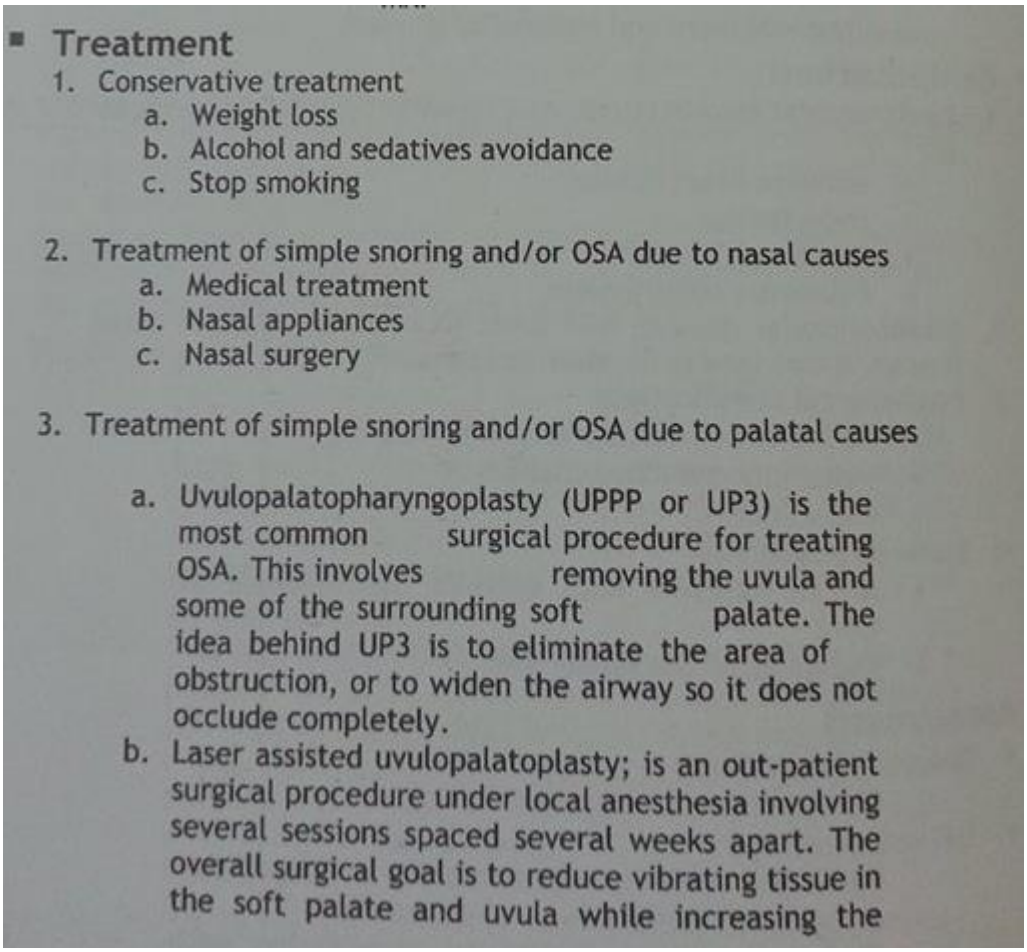
c. Conversion voice disorders (Hysterical voice dis

A conversion reaction is the production of physical
underlying pathophysiological disease, if the larynx is c
the conversion reaction it can result in muteness aphonia
are widely open, full abduction, but contracts and addu
indicating absences of organic paralysis.

d. Mutational falsetto (puberphonia)

Mutational falsetto is the failure to change from a pr
voice to the lower pitched voice of male adulthood. It
of the larynx or vocal cords. Occasionally, chronic disc
asthma, may prevent or retard laryngeal development

17. Treatment of snoring and OSA.(3)



- **Treatment**
  1. Conservative treatment
     a. Weight loss
     b. Alcohol and sedatives avoidance
     c. Stop smoking

  2. Treatment of simple snoring and/or OSA due to nasal causes
     a. Medical treatment
     b. Nasal appliances
     c. Nasal surgery

  3. Treatment of simple snoring and/or OSA due to palatal causes

     a. Uvulopalatopharyngoplasty (UPPP or UP3) is the most common surgical procedure for treating OSA. This involves removing the uvula and some of the surrounding soft palate. The idea behind UP3 is to eliminate the area of obstruction, or to widen the airway so it does not occlude completely.
     b. Laser assisted uvulopalatoplasty; is an out-patient surgical procedure under local anesthesia involving several sessions spaced several weeks apart. The overall surgical goal is to reduce vibrating tissue in the soft palate and uvula while increasing the dimensions of the airway at the retro palatal level.
     c. Palatal stiffening operations

  4. Treatment of severe OSA
     - Continuous positive airway pressure CPAP: usually is administered at bedtime through a nasal mask. The mask is connected by a tube to a small air compressor. The CPAP machine sends air under pressure through the tube the mask, where it imparts positive pressure to the upper airways. This essentially "splints" the upper airway open and keeps it from collapsing.
     - Tracheostomy

18. Congenital anomalies of the larynx.(2)(دي محاضرة كاملة -_-)